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ANNALS
OF
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VOL. 61 SEPTEMBER, 1952 NO. 3

LIII

PAPILLARY SINUSITIS

A. F. JUDGE, M.D.

MARTINSBURG, W. VA.

Papillary sinusitis is a term which rarely appears in the literature. This is due to three factors: The rarity of its occurrence, the confusion in terminology and the differences in opinion as to whether the condition, under its various names, is an inflammatory hyperplasia or a true new-growth. It is the purpose of this paper to try to clarify some of the confusion regarding this type of sinusitis.

Table 1 shows the classification of chronic sinusitis on a basis of morphology as described by Schall,[1] Asch[2] and Eggston.[3] Eggston is the only one to classify papillary sinusitis as a pathologic entity.

Fig. 1 is an illustration taken from the 1942 *Atlas of Otolaryngic Pathology*.[2] It shows a polypoid lesion in which the epithelium has undergone metaplasia to transitional and squamous type. The epithelium is well confined to the basement membrane. This is designated as an "epithelial polyp." This same specimen had been diagnosed by Ewing as an "epithelial papilloma."

Fig. 2 is an allustration from a text on *Pathology of Tumors* by R. A. Willis, 1948.[4] This shows nasal epithelium which has un-

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From the Department of Otolaryngology, Veterans Administration Center, Martinsburg, West Virginia.

This paper was reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

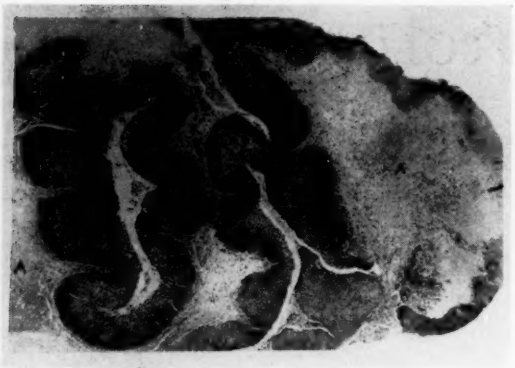


Fig. 1.—From *Atlas of Otolaryngic Pathology*, 1942. Shows stratified squamous epithelium in a lesion diagnosed as epithelial polyp of the nose. This same lesion had been diagnosed by Ewing as epithelial papilloma of the nose.

dergone metaplasia to a squamous type. This has been designated as a "soft or diffuse papilloma."

Fig. 3 is taken from Eggston's *Histopathology of the Ear, Nose and Throat*[3] and is designated as Papillary Sinusitis, epidermoid type.

A careful comparison of the illustrations will show that we have one pathologic entity appearing under four different designations: namely, (1) Epithelial polyp, (2) Epithelial papilloma, (3) Diffuse or soft nasal papilloma, and (4) Papillary sinusitis. Herxheimer and Zaranza have also suggested the term fibro-epithelial polyp.

Billroth[5] in 1883 first described the condition as soft papilloma of the nasal mucosa which he regarded as neoplastic. Hopman, Kiesselbach, Hellmann and Heyman subsequently described this papillary condition of the nasal and sinus mucosa as a benign neoplasm.

Lacoarret and Moure in 1889 asserted that the condition represented hypertrophy of the nasal and sinus mucosa rather than a new-growth.

Wright[6] subsequently called attention to the difference in terminology for the same pathologic condition.

In 1935, Kramer and Som[7] reviewed 86 cases of true papilloma of the nasal mucosa. These included 32 cases of diffuse papilloma

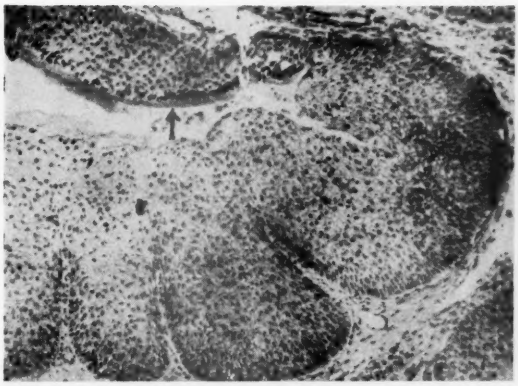


Fig. 2.—From *Pathology of Tumors* by R. A. Willis, 1948. Showing ciliated respiratory epithelium and stratified squamous epithelium in a lesion diagnosed as nasal papilloma.

arising from sinus mucosa. They continued to regard the condition as neoplastic.

Hall[8] in 1937 described soft papilloma as an inflammatory hyperplasia of the mucosa.

Ringertz[9] in 1938, in describing tumors of the nose and sinus cavities, stated that these diffuse papillomas should not in reality be called papillomas. If the term were to be continued in use, then their inverted papillary structure should be stressed.

However, such eminent pathologists as Ewing[10] and Willis[4] continue to list these diffuse papillomatous structures as tumors.

Eggston[3] in 1947 gave a comprehensive description of papillary sinusitis and justified his conclusion that the condition should be regarded as inflammatory in nature.

Most recently, Lehman[11] in 1949, has added a case of papillary sinusitis to the literature.

INCIDENCE

Kramer and Som[7] in reviewing the literature in 1935 found 86 cases of papilloma of the nasal cavity. Of these, 32 were cases of diffuse type arising in the sinuses which should be considered papillary sinusitis.

Fig. 3.—From *Histopathology of the Ear, Nose & Throat* by Eggston and Wolff, 1947. Shows squamous epithelium in a lesion diagnosed as papillary sinusitis, epidermoid type.

Ringertz[9] in 1938 presented 11 cases which he had encountered. He believed that occurrence was more frequent but diagnosis was not made because the lesion frequently resembled ordinary nasal polyps.

Nigro[12] in 1949 presented a study of 200 cases of sinus surgery in which the sinus mucosa was studied microscopically. Two of these were found to be papillary sinusitis.

Table 2 shows the incidence of papillary sinusitis encountered in the routine examination of sinus mucosa removed surgically at the Manhattan Eye and Ear Hospital for the past three and one-half years.

Table 3 shows the incidence at the Veterans Administration Center, Martinsburg, W. Va., for the same period.

From the foregoing, it would appear that when sinus tissue removed surgically is routinely examined, the incidence of papillary sinusitis is about 0.8%.

All authors agree that the disease is found more frequently in males than in females and in the 40-50 year age group.

LOCATION

Kramer and Som[7] found the ethmoid sinus most frequently involved and the antrum next.

Eggston[3] found the antrum and ethmoid to be most frequently involved.

Ringertz[9] found the ethmoid sinus to be most frequently involved.

Lobel found one case in which the process had spread to the spheno-ethmoid region.

Lehman[11] reported one case in which the frontal sinus was involved.

In two of our cases the process involved the frontal sinus by extension from the ethmoid.

Wallner[12] has described lesions with similar papillary changes in the mucosa appearing on the posterior ends of the middle turbinates.

ETIOLOGY

Eggston[3] believes the process to be due to a virus infection. This belief is based on the morphologic characteristics and its diffuse spread over the mucous membrane. He feels that the presence of irritating and degenerating proteins associated with inspissated inflammatory exudate in the sinuses tends to cause papillary growths.

Kramer and Som[7] noted that a long history of infection was usually associated with the condition. However, they felt that sinus infection was too common and papillary sinusitis too rare to establish any etiologic relationship.

Ringertz[9] felt that papillary changes might develop in preexisting polyps. From examination of his material, he felt this was a fairly common occurrence.

Kern and Schenck[13] have called attention to the close relationship between nasal polyps and allergy. These polyps often show areas of metaplasia of epithelium. Wallner has shown that a large percentage of patients with mulberry hypertrophy of the inferior turbinate have a history of allergy. Both of the author's cases of papillary sinusitis had a definite history of allergy. The relatively common occurrence of nasal polypi and allergic states as compared to the rarity of papillary sinusitis would rule out allergy as a single etiological factor. However, it seems to the writer, if we consider the existence of a basic allergy and then have a superimposed virus infection, this might acount for the appearance of a papillary sinusitis.

PATHOLOGY

Grossly, the condition may be manifest in the nose as polyps which, however, do not have the smooth surface of the usual edematous polyp, but have an irregular, velvety appearance. They are more red and bleed more easily than mucous polyps. In the nose or in the sinuses they extend over a wide area of mucous membrane.

Microscopically, there is a metaplasia of epithelium and an inward growth of the epithelium forcing the stroma apart. This results in the formation of crypts in which the epithelium may appear as stratified cylindric epithelium. There is always a definite basal membrane. The stroma is loose and rich in blood vessels. Inflammatory cells are scattered throughout the stroma. In addition to metaplasia, the epithelium also undergoes papillary hyperplasia. Eggston[3] has described papillary sinusitis of an epidermoid, foliate, cryptic, and filiform types depending on the predominating morphology.

Bacteriological examination of the specimen reveals staphylococcus, nonhemolytic streptococcus, and micrococcus catarrhalis which are the usual organisms found in the nose.

SYMPTOMS

There are no specific symptoms of the disease. Depending on the location of the papillary growths, they may cause nasal obstruction, loss of smell, and nasal discharge. If there is pressure necrosis of bone the disease may cause orbital symptoms. Nasal examination usually reveals a red exuberant tissue elevated above a surrounding mucosa which appears normal. Mucus or pus may be present depending upon the coexistence of suppuration.

X-rays usually show a glossy, homogenous opacity of the involved sinus. This can rarely be distinguished from polyposis. Erosion of bone may be present due to pressure and not to actual invasion. One of the author's cases showed erosion of the roof and medial wall of the orbit.

PROGNOSIS

Hellmann, Poli, and Holmgren reported the tendency of this condition to recur. Of Ringertz'[9] eleven cases, four showed recurrence and one eventually became malignant. Of the five cases which Kramer and Som added to the literature, one changed to malignancy. They also noted a tendency to recurrence.

Among the cases followed for a number of years at the Manhattan Eye and Ear Hospital, Eggston[3] found no recurrences. One case, however, became malignant.

The writer's two cases have been followed for too short a period to draw any conclusions.

TREATMENT

Because of the recurrent characteristic of the disease, Kramer and Som recommended total removal by surgical diathermy followed by irradiation.

The cases at the Manhattan Eye and Ear Hospital were usually treated by radical exenteration.

Ringertz[9] emphasizes that all nasal polyps should be examined histologically as they occasionally are found to be undergoing metaplasia of the epithelium. He noted greater frequency of nasal polyposis in patients with nasal cancer developing from a papillary type of membrane than the average frequency of polyposis in his whole cancer material. His cases were treated by electrosurgical excision and at times followed by radium application or irradiation by x-ray. On occasion, the recurrence responded poorly to irradiation.

Recently, Holinger, Johnston, and Anison[14] have reported encouraging results in the treatment of papilloma of the larynx by supplementing surgery with the use of aureomycin. Hollingsworth, Kohlmoos, and McNaught[15] have reported good results in the same disease by the local use of resin of podophyllum. Estrogenic hormones have also had some salutary effect in laryngeal papilloma. There is a great deal of similarity between juvenile papilloma of the larynx and papillary sinusitis, so it is possible that the above measures might be useful adjuvants in the treatment of papillary sinusitis.

The author's patients were treated by radical external frontal and ethmoid sinusotomy. Aureomycin was used post-operatively, locally and by mouth. The allergic factor was controlled by antihistamine.

REPORT OF TWO CASES OF PAPILLARY SINUSITIS

CASE 1.—Z. R., a sixty-four-year-old, white, male, was admitted to the Veterans Administration Center because of difficulty breathing through the right side of the nose and profuse purulent nasal discharge of eight years' duration. Patient also complained of occasional pains in the right frontal region. He had suffered from

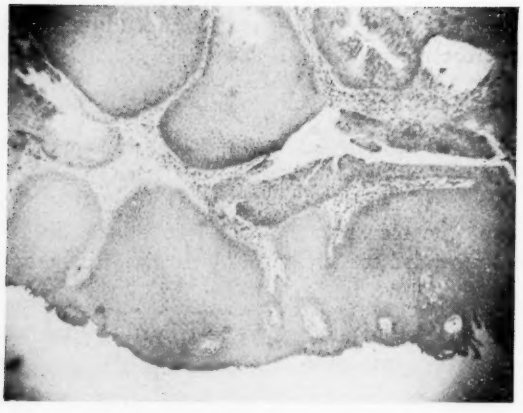
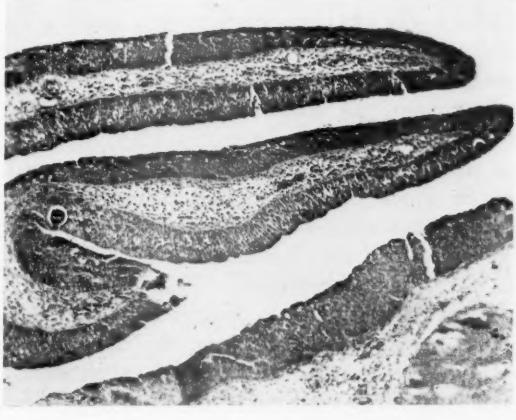


Fig. 4.—Shows squamous epithelium in the mucosa removed from the ethmoid sinus of Case 1.

hay fever and eczema of the ears for over twenty years. Examination showed the right side of the nose to be filled with mucopus and large, red polyps filling the right middle meatus. There was moderate desquamation of epithelium of both external auditory canals. Laboratory data: Blood hemogram was normal; urinalysis was normal; blood Kahn was negative. X-ray examination of the paranasal sinuses showed diffuse clouding of all the sinuses on the right side and a lack of detail in the floor of the frontal and the lateral wall of the ethmoid sinus suggesting erosion of bone.

Course in Hospital: On April 10, 1950, under local anesthesia, the nasal polyps were removed by snare. Histological examination showed the epithelium of the polyp to have changed to stratified squamous epithelium with inflammatory infiltrate in the stroma and cyst-like structures being numerous. The lesion was considered pre-cancerous. Following removal of the polyps and the institution of penicillin and antihistamine therapy, the nasal exudate largely disappeared. On May 11, 1950, under general anesthesia, a right radical frontal and ethmoidal sinusotomy was performed. Three areas of erosion of bone were found in the roof and medial wall of the orbit. The membrane throughout the frontal and ethmoid sinuses appeared thickened and polypoid and mucopus was present. Bacteriological study of the exudate showed staphylococcus aureus hemolyticus and alpha streptococcus. Post-operatively the penicillin and antihistamine were continued and in addition the patient re-



Figs. 5 and 6.—Show details of the epithelium of mucosa removed from the frontal sinus in Case 2. X 10 and X 48 respectively.

ceived a total of 7000 mg of aureomycin by mouth. Convalescence was uneventful. The microscopic diagnosis of tissue removed from the sinuses was hyperplastic papillary sinusitis. At the time of last examination, there was no evidence of recurrence.

CASE 2.—O. J. P., a fifty-seven-year-old, white, male, was admitted to the Veterans Administration Center complaining of difficulty breathing through the nose for several years. He had suffered from nasal discharge which tended to crust during the same period. He stated that he had always suffered from hay fever, as had one grandfather. In 1930 he had been treated for gonorrhea and syphilis. In 1935 he received malaria treatment for neurosyphilis. Examination showed mucopus present on both sides of the nose and polypoid tissue anteriorly in the right middle meatus. Laboratory data: Blood hemogram was normal; blood Kahn was negative. Examination of spinal fluid showed an increase of globulin, a positive serology, but a negative gold curve. X-ray examination showed clouding of the right frontal and ethmoid sinuses with thickening of the cell walls. The left maxillary sinus was also cloudy.

Course in Hospital: Because of the spinal fluid findings, the patient was given 6,000,000 units of penicillin over a period of ten days. On March 20, 1951, under local anesthesia, a right frontal and ethmoidal sinusotomy was performed. The membrane was found to be polypoid and the intracellular septa were atrophic. Mucopus was present. A culture taken at the time of operation showed an alpha hemolytic streptococcus, staphylococcus albus and micrococcus catarrhalis. Histologic study of the removed membrane showed areas in which the epithelium had undergone metaplasia to a stratified squamous type with papillary formation. There were numerous inflammatory cells present in the metaplastic epithelium and also in the supporting stroma. Post-operatively, the patient received aureomycin by mouth as well as antihistamine drugs. On April 10, 1951, under local anesthesia, the membrane of the left antrum was removed by means of a radical antrotomy. This membrane showed only chronic inflammatory changes without squamous metaplasia. Convalescence was uneventful. To date, there is no evidence of recurrence of the papillary sinusitis.

SUMMARY AND CONCLUSIONS

1. From a study of photomicrographs it is seen that papillary sinusitis appears in the literature under various appellations; e.g., epithelial polyp, epithelial papilloma, diffuse or soft nasal papilloma, fibro-epithelial polyp.

2. The condition is rare. In routine examination of surgical specimens of sinus mucous membrane at the Manhattan Eye and Ear Hospital and a Veterans Administration Center over a period of three and one-half years, papillary sinusitis was encountered nine times in twelve hundred specimens.

3. Most writers stress the tendency of the condition to recur and its precancerous nature. Eggston disagrees with both these postulations. However, clinical experience indicates it should be regarded with suspicion.

4. If one accepts the possibility of virus infection as the etiology, the use of aureomycin to supplement surgery may prove efficacious.

5. Two additional cases are reported.

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LIV

NEURILEMMOMA OF PHARYNX

LIEUT. COL. KENNETH SOMERS

DENVER, COLO.

I have found recorded in the literature 29 cases of neurogenic tumors occurring in the pharynx and derived from elements of the peripheral portions of the nervous system. There was one plexiform neuroma,[1] one ganglioneuroma,[7] and 27 neurilemmomas. Two of the neurilemmomas reported by Koop[13] had ganglion cells present in the tumor, so that they might have been classified histologically by some pathologists as ganglioneuromas. The one case of plexiform neuroma could have been classified as a ganglioneuroma for Doctor L. H. Meeker, a pathologist, in discussing Forbes' paper[1] stated: "Clinically this is a plexiform neuroma, but microscopically this is a true ganglion neuroma." Doctor W. J. MacNeal in discussing the histological picture of this same tumor stated, "A specimen of this sort indicates that the peripheral nerve tumors are essentially one group and not distinctly separated into ganglionic neuromas and neurofibromas."

Neurilemmoma has been called by numerous names by different authors such as solitary neurofibroma, neurinoma, fibroma of nerves, gliofibrosarcoma, peripheral glioma, Schwannoma;[2] perineural fibroblastoma, neurilemoma,[3] neurilemmoma;[8] neuroma;[14] perineural fibroma and neurogenic fibroma.[15]

Ganglioneuroma has been called ganglionar neuroma, ganglionated neuroma, ganglionic neuroma;[16] ganglion cell neuroma and ganglioma.[15]

The plexiform neuroma has few synonyms and it is described by Anderson[17] in a vivid way, "A tangle of well differentiated hypertrophied peripheral nerve filaments resembling grossly a tangle of spaghetti—numerous well differentiated nerve bundles including all adult parts."

It can be said, without going into details of the microscopic picture of these peripheroneurogenic tumors of the pharynx, that the histological picture of the neurilemmoma is characterized by the

From EENT Service, Fitzsimons Army Hospital, Denver, Colorado.

Schwann cell, the ganglioneuroma by the ganglion cell and the plexiform neuroma by nerve bundles. In the last analysis however, as stated by Hall,[5] "The diagnosis in a case of this type can be made only by the pathologist, and even he admits that there may be room for controversy over the microscopic picture."

Even as late as 1947 Orton[12] stated that neurofibroma was one of the rarest tumors of the pharynx and in 1941 Hall and Owens[5] stated that it was, "one of the rarest tumors ever to be encountered by a laryngologist." Stout[3] in 1935 in speaking of neurilemmoma said the tumor had never been found in the feet, the genitourinary system, the lungs, the esophagus or the rectum. Perhaps neurilemmoma is not so rare nor so confined as to occur in only certain locations as was thought a decade or two ago. Norris and Cooper[18] have reported a neurilemmoma in the vagina, Engleking, et al,[19] have reported one in the esophagus, Mintz[20] one in the urinary bladder, and Parker[21] one of the vegetative nervous system.

Of the 29 cases of peripheroneurogenic tumors of the pharynx found in the literature I found 19 cases sufficiently well described to be included in the table. Nine cases were reported by New and Devine[15] which were not described in enough detail to be included in the table and I was unable to consult the article by Askanazy[22] describing one case. Since clinically the plexiform neuroma and the ganglioneuroma appear to behave similarly to the neurilemmoma there seems to be no point in separating these three types of tumors when consulting the data in the table.

The ages range from 14 to 61 and are distributed mostly through the third to the sixth decade. Males and females are about equally divided.

The largest tumor dimensions given in specific units of measurement was from the case described by Figi[2] and is 8 x 6 x 4.5 cm. The smallest tumor dimensions are reported by Stout[3] and are 3 x 2 cm. The average size of all tumors for which definite measurements were given was 5.1 x 3.7 x 2.6 cm.

There were 17 of the 19 tumors removed surgically and of these 17 there was some mention made of the amount of bleeding during operation in 9 cases. In only 1 case was the bleeding described as more than minimal. It can be assumed that in the 8 cases in which bleeding was not mentioned it was insignificant, otherwise it probably would have been mentioned. In spite of the bleeding being minimal the external carotid artery was ligated in two instances, the superior thyroid and lingual vessels in one instance,

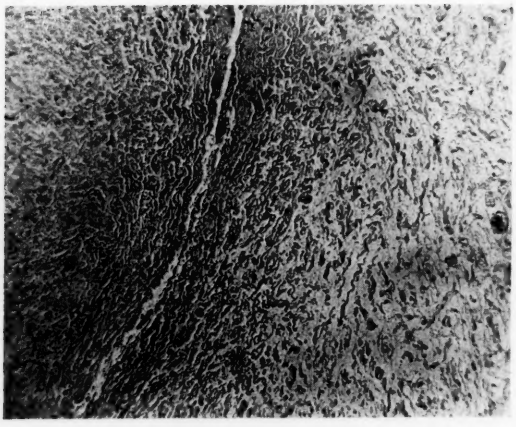

Fig. 1.—Histological structure of tumor, 90X.

and the internal carotid artery once, this latter case resulting in death. It would appear from the records that pre-operative ligation of large vessels is seldom necessary.

The surgical approach has been about four times more frequent through the mouth than by pharyngotomy. Only two cases were stated to have been operated on under local anesthesia.

The anatomical site of the tumor has in most instances been either lateral to or behind the tonsil and the right and left sides are about equally divided. All cases except one of those reported by Ehrlich and Martin have been described more to one side than to the other, Ehrlich and Martin's case being described simply as retropharyngeal. The case which I will report was almost in the midline but perhaps somewhat to the left side.

The postoperative sequelae have been few and mostly due to temporary or permanent paralysis of the cervical sympathetics, or the ninth, tenth or twelfth cranial nerves or their branches.

The main symptoms have been difficulty in swallowing or knowledge of the physical presence of a mass. The symptoms have been present from 2 weeks to as long as 18 years before removal of the tumor.

CASE REPORT

The patient was a 20 year-old-male admitted to Fitzsimmons Army Hospital on June 21, 1950 as a transfer from another hospital

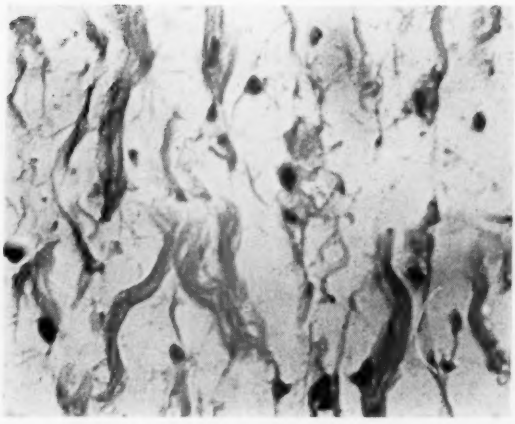

Fig. 2.—Histological structure of tumor, 450X.

with the presumptive diagnosis of cervical tuberculosis. The initial onset of left cervical adenopathy was in 1943. A biopsy was done at that time in a civilian hospital and the patient was told he had a "fibrous tumor." He was asymptomatic until June 1948 when he developed a sore throat and swelling of the left side of the pharynx, and painful swelling on the left side of the neck just distal to the site of the old operation. This was accompanied by a low grade fever and malaise. He was hospitalized several weeks, had many cervical and chest x-rays and when sent to duty he was not told what diagnosis had been made. In January 1950 he was admitted to another hospital because of low grade fever, malaise, sore throat and difficulty in swallowing. Physical examination was negative except for bulging of the pharynx on the left side, and cervical swelling below the left mandible. The roentgenologist reported a mass in front of the cervical spine which was thought to be a retropharyngeal abscess. A trocar was inserted into the area but no purulent material was obtained. A biopsy was obtained from the cervical lymph nodes and was reported as chronic lymphadenitis.

On admission to this hospital the physical examination was essentially negative except for the mass in the back of the pharynx and a submandibular swelling. After clearance by the tuberculosis service the patient was operated on and the pharyngeal mass removed in October 1950.

The operation was performed under pentothal sodium anesthesia. The approach was through the mouth and the tumor shelled out easily by blunt dissection with only minimal bleeding. A biopsy specimen was taken from the left cervical swelling at the same operation and histologically showed only reactive hyperplasia. The patient made an uneventful recovery.

The pathologist's report on the gross specimen was as follows: "Specimen consists of five irregular pieces of tissue, the largest measuring 3 x 2 x 1 cm. Here and there on the surface of this tissue there are round, fairly white masses which on sectioning are solid throughout. The remaining tissue was friable varying in color from light yellow to dark brown."

The report on the microscopic appearance was as follows: "Consists of circular nodules which have an indefinite surrounding capsule derived from the surrounding fibrous tissue. These nodules show a loose network of somewhat fragmented fibers with considerable edema between them. In some, these fibers are more compact and show a whorled arrangement. In other areas, the ground substance is more amorphous than fibrillary. This fibrillary material contains nuclei which are either oval or spindle shaped and the chromatin is homogeneous. The appearances are those of a benign neurofibroma." A representative microscopic field is shown in Figures 1 and 2.

SUMMARY

A review is made of 29 peripheroneurogenic tumors occurring in the pharynx. The great majority of these tumors were neurilemmomas.

A case report is given of a neurilemmoma of the pharynx which was associated with submandibular swelling with a resulting diagnosis of retropharyngeal abscess at one time and of cervical tuberculosis at another time.

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LV

LARYNGOLOGY AND VOICE PRODUCTION

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Ever since the day when Garcia made his first laryngeal examination and succeeded in observing some two thirds of his vocal cords, the larynx has been the subject of the most careful and detailed study. Since it is due to the action of the vocal cords that vocal tone is produced, efforts have been constantly made to solve all vocal problems by relating them to the action of the vocal cords. This is quite understandable for the larynx was considered to be the "vocal organ" and it was believed that it was designed expressly for the purpose of producing tone. This attitude has given way to the realization that despite the fact that the larynx produces tone, its original and most important function was that of keeping food and liquid out of the lungs. Up till now, this has been regarded as somewhat of an academic point and its full significance has not been appreciated. However, once this fact has been accepted we find ourselves using an organ for a purpose for which it was not specifically designed. If man had not discovered that it was possible to make sounds by means of this valvular organ, and had continued to make use of it solely as a means to prevent food and liquid from entering the lungs, most of the disabilities which afflict it, would never have come into being. I refer of course to those disabilities which have their origin in the use of the larynx for tone producing purposes.

Nature has been so expert in so many of her designs that had she found it necessary to design a tone producing organ fitted for this one purpose, the study of voice production would have been largely unnecessary, and aside from pathologic conditions, it would have always functioned properly.

So many vocal disabilities can be traced to improper use of the larynx that the time is long over-due for a thorough re-examination of the premises which both laryngologists and vocal teachers have accepted.

The author, a teacher of singing, was trained as an engineer. He has contributed articles on voice production to various journals.—Ed.

One of the most important points which must be considered is what constitutes "improper use of the voice?" And again, why do many people go through their whole lives hardly conscious of the fact that they possess a larynx, while others find themselves often in difficulties which are sufficiently serious to warrant consultation with a laryngologist? When we consider those who use their voices professionally the situation is far worse. Why does one public school teacher find that she has developed vocal nodules, whereas her colleagues are able to maintain an even heavier schedule than she does without any such difficulties? Why does one singer find herself forced to relinquish what seemed to be a promising career, whereas another is able to continue singing for a far greater period of time? These are questions which must be answered by those whose profession it is to train, and by those whose profession it is to treat the vocal organ it becomes adversely affected.

One of the most frequently encountered vocal disabilities are vocal nodules. Not only do they affect singers, most frequently women, but also others, such as teachers and those who have to use their voices to a greater extent than would be entailed in ordinary conversation.

Nodules are almost invariably to be found located at the anterior third of the vocal cords and are recognized as being caused by excessive irritation of the free edges of the cords.

In normal voice production the cords are actuated by a group of muscles commonly referred to as the intrinsic muscles of the larynx, since, with the exception of the cricothyroid, the palatopharyngeus and stylopharyngeus, they are located inside the larynx. These are the thyroarytenoids, the lateral cricothyroids and the posterior arytenoid. The posterior arytenoid approximates the arytenoid cartilages, tension being induced longitudinally by the thyroarytenoids and the lateral cricothyroids. Externally the cricothyroids, the palatopharyngeus and stylopharyngeus exert a similar pull on the cords, the latter two muscles accomplishing this by tilting the thyroid cartilage forward.

If the actions of this group of muscles be carefully followed, it will be noted that none of these muscles is capable of exerting pressure at the anterior third of the cords where nodules are to be found. It should, therefore, be obvious that since pressure must have been induced in order to provide the necessary irritation which is responsible for the formation of the nodules, this pressure must derive from some source other than that of the intrinsic or vocal

muscles proper. From this it follows that a study of the larynx itself is not sufficient to explain the formation of vocal nodules.

The action of the larynx is subconscious, recognition of this fact is evidenced by the direction which the examiner gives to his patient. He does not say: "approximate your vocal cords" which would be meaningless to the patient, he directs him to say or sing an "E". Prior to this, however, the patient is directed to put out his tongue, a recognition that the tongue is under conscious control.

The problem which faces the investigator is, how can an organ, not subject to conscious control, develop a disability which cannot be brought about by the action of its own muscles? How can a lesion of the free edges of the vocal cords develop when the action of the muscles which control the vocal cords precludes any such possibility?

To understand how such an anomaly can occur it is necessary to return to the premise that this organ, designed primarily as a valve to prevent the ingress of food or liquid into the lungs, is being used for a purpose other than for which it was originally intended. Its valvular action is brought about not alone by its own muscular system, but also by the muscles of deglutition, notably those of the tongue. This combined muscular action causes a sphincter action to occur. Obviously an attempt to phonate during a complete closure of the larynx would fail utterly, but, if a partial stimulation of the muscles of deglutition be induced, phonation becomes possible. With the aid of the powerful abdominal muscles it is possible to drive air through the partially closed larynx and to produce tone, but such voice production will inevitably react unfavorably upon the vocal organ.

While this type of forced production is fairly common among singers it is also to be found, though to a lesser degree, among those who use their voices solely for speech. But the results, so far as the larynx is concerned, are always unfavorable. As has already been emphasized, it is this failure to connect the action of the swallowing muscles with phonation which has prevented researchers in vocal problems from obtaining a clear picture of what can, and often does take place when a person phonates. Obviously laryngoscopy would reveal nothing of this, the fact that the tongue is drawn forward during the examination would prevent the observer from gaining any knowledge as to what takes place when the tongue is free to be drawn down upon the larynx during forced voice production. How can it be thought possible that the laryngologist could gain a clear picture of the causes which result in the formation of nodules when

his observations are limited to the laryngoscopic image? This misapprehension is unquestionably due to the concentration of research upon the larynx and a more or less complete failure to relate the effect of the actions of other muscles of the throat upon the vocal organ. It is responsible for an actual impasse and limits the value of the laryngologist to his patients.

In the majority of cases of vocal disability the patient is advised to rest his voice, and certainly complete rest will benefit almost any vocal condition, but this can be regarded only as a palliative. After sufficient rest a complete recovery may be noted, but when the patient begins to use his voice again, the old trouble will recur. Under present conditions the laryngologist is barred from providing real help to such patients for he is dealing with the larynx itself and the origin to the trouble lies above the larynx. This area is considered out of bounds so far as he is concerned, it supposedly belongs to the voice expert.

Voice experts are usually recruited from the ranks of professional singers and musicians, who, while they may have some practical knowledge of music, are, generally speaking, completely ignorant of physiology. Still believing that the vocal organ is a musical instrument, they treat it as one, seldom seeming to realize that it is an integral part of the human body. It is a situation in which only the toughest survive what passes for vocal training. The weaker ones simply fall by the wayside.

At this point it becomes possible to answer the question put earlier, namely, why it is that some individuals experience greater difficulty in producing their voices, professionally or otherwise, than others? This depends entirely upon the degree of tension exerted by the swallowing muscles upon the vocal organ. This varies greatly, some people producing their voices with a minimum of this external tension, whereas in others it exists to so great a degree as to render voice production not only difficult, but in extreme cases, impossible. Production of vocal tone with the hampering effect of this external tension allows the true vocal muscles less and less freedom of action, consequently depriving them of the exercise necessary to keep them in good condition. How necessary this exercise is was emphasized by a statement made many years ago by Morrell-Mackenzie. He said: "A singer's thyroarytenoid muscles should be in as good condition as a pugilist's biceps.

From time to time there have been suggestions made that the teachers of voice production and the laryngologists should cooperate with one another, but so far, little, if anything, of this sort has been

accomplished. Each goes his own way and they meet only when a pupil's larynx rebels against the treatment it is receiving and refuses to function any further. Then medical aid is sought. Again rest and possibly some medication will be prescribed, and in due time the pupil returns to his work and almost inevitably to a recurrence of his troubles.

It is of the greatest importance to singers and speakers that this vicious circle be broken. To accomplish this it would almost seem necessary for laryngologists to become voice teachers and voice teachers, laryngologists. Obviously such a solution would hardly be feasible, but certainly a pooling of information which has been acquired by both professions is long overdue.

In the "Journal of the American Medical Association" (November 3rd, 1928) Dr. Elmer L. Kenyon drew attention to the effect of the extrinsic throat muscles upon speech and outlined a method of diagnosing vocal difficulties by means of finger palpation. By placing the finger above the larynx and observing its movements, he pointed out that it was possible to learn whether the internal muscles were being occorded freedom of action. With slight variations this principle has been employed by me to diagnose and eliminate faulty habits of song and speech for nearly forty years. In "The Laryngoscope" (July, 1935) I stated that vocal nodules could be cured by the use of this method of finger palpation. In "The Laryngoscope" (November, 1939) I again showed how various other vocal disabilities could be overcome by the method of finger palpation. In the "Archives of Otolaryngology" (November, 1948) I reported upon several cases involving speech and also how an opera singer's troubles were cured by this same procedure.

The use of the fingers supplies the much needed desire for something definite upon which to build. The manner in which this is accomplished is relatively simple. With the finger placed in the angle of the patient's jaw (above the larynx) the patient is directed to swallow. The considerable muscular contraction which take place should be carefully noted. Then, with the finger in the identical position the patient is directed to sing or speak. If this is found to be impossible without a similar though less pronounced muscular contraction occurring, the patient is obviously involving the swallowing muscles. Even a very slight tensing of these muscles will have an adverse reaction upon the patient's voice production.

With so simple and reliable a method of diagnosing vocal difficulties available it can only be regretted that laryngologists and vocal teachers have not taken advantage of its benefits.

Another test which unfailingly supplies evidence of undue force in the production consists of the following procedure. This would apply specifically to singers for speakers do not have occasion to make use of sustained tones or tones of a relatively higher pitch. With the finger in the same position the patient is directed to sing a tone in the upper range of his voice. The tone should be started very softly, then the volume increased and following this to return to the original small volume. If this is difficult or impossible to accomplish this test supplies evidence that the tone production is faulty.

As has been mentioned earlier, it is quite usual for singers to produce tones with the aid of the swallowing muscles. This type of voice production requires a considerable amount of breath pressure in order to force the cords apart. This results in the production of a tone of considerable volume. The attempt to reduce this volume almost always causes a sudden cessation of the tone. With a lessening of the breath pressure the vocal cords will be found to close with the result that phonation becomes impossible. Correct voice production permits the change in volume to be achived with an almost imperceptible increase in the amount of breath used, and when the voice is being freely produced, no involvement of the external muscles can be detected.

It need hardly be said that this outline represents only the beginning in the search for an improvement in methods of diagnosing faulty habits of voice production, but with care and persistence the investigator will soon find himself able to point out to his patients or pupils how some of the many existing pitfalls can be avoided. Voice production is not an esoteric mystery to which only a chosen few have the key. It is merely the following out of simple rational procedures and practices such as are to be found in all matters connected with psycho-physical skills.

LVI

METHOD OF SHAPING A PLASTIC IMPLANT FOR RECONSTRUCTION OF THE NASAL BRIDGE

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The purpose of this note is to suggest a method of making implants for rhinoplastic surgery which fit, remain anchored and have a measure of mobility. Numerous materials have been used as grafts in rhinoplastic surgery for the reconstruction of the nasal bridge, but none of these has been entirely satisfactory. Plastic materials are now being used in eyelid reconstruction work, in orbital implants, as well as in rhinoplastic surgery. If experience shows these materials to be more suitable, implants made from them offer certain advantages, chief among which is the accuracy with which they can be shaped before operation, by standard dental methods. In addition, they are more or less mobile and they remain in place. The wax model of the implant may be shaped in the surgeon's home, at his leisure, and the implant processed in a standard dental laboratory.

Technic. A wax model of the patient's face is made in the usual manner and pure dental tinfoil 1/1000th of an inch in thickness is burnished into position over the general area to be covered by the implant (Fig. 2). A model of the implant is then built from colored dental inlay wax, using a standard dental spatula, heated for adding wax, and carving it. The line of the bridge, as seen in profile, is built up exactly as desired because when the implant is slipped under the skin at operation, it takes a position which is posterior to the extent of the thickness of the skin. However, if the upper limit of the implant lies about midway on the bony portion of the bridge, it must be cut down somewhat, otherwise this end may lever up, lifting the skin, because the lower end over the soft cartilaginous portion of the nose may sink. If the upper end of the implant lies inferior to the nasal bones, this precaution is not necessary, neither is it necessary if it extends to the upper end of the bridge and lies against the glabella. From side to side, the implant must be narrower than the reconstructed nose, otherwise, when it is placed in position, the nose will be wider, by twice the thickness of the skin, than it should be and the implant may fit much too tightly. If the implant

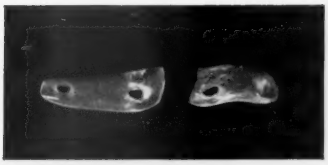

Fig. 1.—Two portions of an implant received from the laboratory after polishing, showing the perforations through which cords of connective tissue are expected to form.

is to come down materially on the sides of the nose, some of the wax on the face model must be carved away so that it can be properly narrowed. It is best not to be too zealous in bringing the outline to sharp feather edges because these make for rigidity, immobility and may irritate adjacent tissue; the margins should be round rather than sharp.

When the wax model of the implant has been completed to the operator's satisfaction, it is then divided into two segments, to form an articulation between the portions which lie on the mobile lower third of the nose and the rigid upper two-thirds. The line of division is located on an arc by means of a compass, one point of which is fixed at the glabella and the other point about at the junction of the middle and lower third of the nose. The cut can be made by the point of the compass or by a small knife or by the wax spatula. When the division is completed, there should be a space for articulation (perhaps 1 to 2 mm) between the two segments of the wax model of the implant.

The two portions of the wax model of the implant are sent to a dental laboratory and processed in methylmetacrylate. When the rough implant has been received from the laboratory, the surgeon perforates it with a dental bur at two or more points, selecting the thinnest spots, and drilling from the posterior aspect. The perforations are widened on the posterior or under side to form very shallow truncated cones, thus eliminating the formation of pockets. The perforations should not disturb the contour of the anterior surface. Both segments of the implant are then returned to the laboratory for thoro polishing in all portions, including the interior of the cone-shaped perforations (Fig. 1). (The theoretic purpose of these perforations is to allow fibroblasts to extend through them, thus uniting the scar tissue at the anterior and posterior surfaces of the implant and anchoring the implant.) When the two portions of

Fig. 2.—Tinfoil burnished into position on the wax model. The model of the implant is shaped in colored dental wax on top of the tinfoil.

Fig. 3.—Profile view of the two portions of the implant in position on the model.

Fig. 4.—Wax model of the same nose some two years after operation. The nose was comfortable, showed no signs of irritation and the tip was mobile.

the implant have been received from the laboratory and laid on the face model, they should appear as in Fig. 3.

The operative procedure itself is so simple as to be almost a minor one. The usual incisions are made at the margins of the alae, extending onto the columella. The skin is separated over the lobule of the nose from the alar (lower lateral) cartilages and a channel made by blunt dissection up along the bridge of the nose as far as desired. The two pieces of the implant are simply slipped into position and adjusted with reference to one another. If correctly prepared, they should drop accurately and loosely into position. (If they fit tightly they may be extruded.) The operation is closed in the usual manner.

When healing is completed, the nose should be without reaction; the lobule should be mobile and there should be no tenderness.

The foregoing is not to be construed as a recommendation for the use of this particular plastic material for nasal implants. Time and experience alone will demonstrate whether it is suitable.

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LVII

BRONCHOGRAPHY IN INFANTS AND CHILDREN

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Brochongraphy in infants and young children has been considered to be a difficult problem by many authors. Eeman[1] and Bates[3] recently reviewed the subject; their review will not be repeated here. The method reported here is a direct outgrowth of that described by Wishart[2] in 1947. It would not have been evolved except for the objection of Minneapolis radiologists to the use of open ether in the fluoroscopic room because of fire hazard. This paper presents a technique applicable even to very young children. This technique has yielded excellent bronchograms in infants and young children and seems to fulfill all safety requirements.

A bronchographic technique for children should satisfy the following criteria: (1) The airway should never be obstructed, and the patient's breathing should always be under the observation and control of those carrying out the bronchoscopy, even in the darkened fluoroscopic room. (2) The anaesthesia should be adequate enough to permit injection of the contrast medium under fluoroscopic control at the will of the operator and without cough or other interference by the patient. (3) The technique should offer no fire hazard. (4) Aspiration of secretions and contrast medium from the respiratory tree should be instantly possible. (5) "Spot film" technique should be available to permit instantaneous recording of bronchographic images.

Complete familiarity with bronchial anatomy and use of a common terminology by the bronchologist, radiologist and thoracic surgeon is essential to adequate bronchial delineation and description of findings.

In our bronchographic studies in children the bronchologist, anesthesiologist and radiologist take part. The technique is as follows:

(1) Anesthesia is induced in the bronchoscopic operating room with the bronchoscopic operating table already set-up to permit instant relief of respiratory obstruction. In some children the larynx

From the Division of Otolaryngology of the University of Minnesota Medical School.

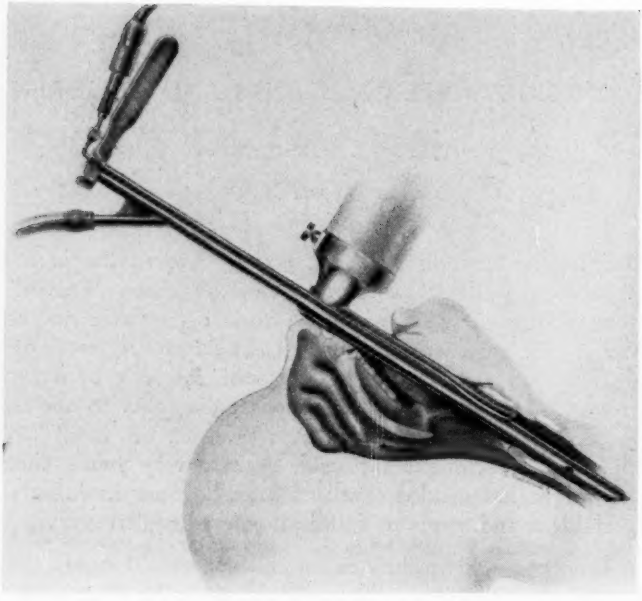

Fig. 1.—The bronchoscope is being introduced through a Flagg laryngoscope with oxygen flowing through the side-tube of the bronchoscope.

is examined and the bronchoscope is introduced before anesthesia is induced. Obstructing secretions can be aspirated and anesthesia induced through the bronchoscope. In other children anesthesia is induced first and is followed by laryngoscopy and bronchoscopy (Fig. 1).

(2) After bronchoscopy is completed a plastic tube is passed through the bronchoscope into the section of the lung to be examined first during bronchography. The bronchoscope is then withdrawn leaving the tube in place. The tube used is such that a number 13 needle makes a tight connection when the tubing is threaded over it. The sharp point of the needle is previously ground off.

(3) Immediately after withdrawal of the bronchoscope the laynx is exposed with the laryngoscope and an endotracheal tube of the ordinary kind used for endotracheal anesthesia is passed through the larynx alongside the plastic tube previously introduced. Thus two tubes traverse the larynx. The endotracheal anesthesia tube is

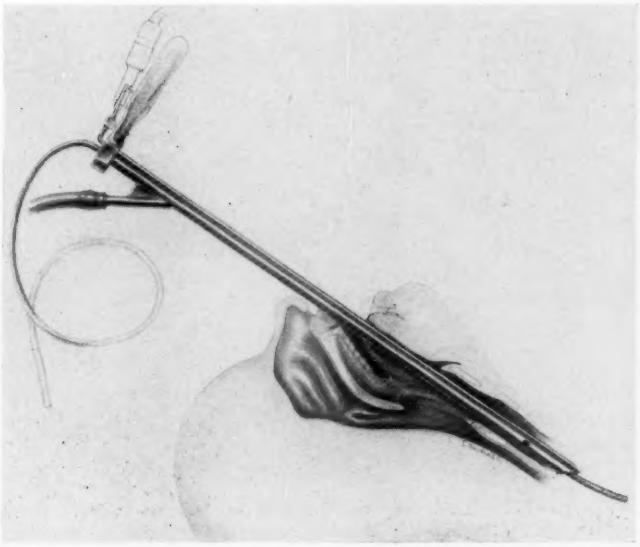

Fig. 2.—The plastic tube for lipiodol injection is being inserted through the bronchoscope into the segment of the lung to be studied first. The bronchoscope is withdrawn leaving the plastic tube in place in the bronchial tree and protruding through the mouth.

immediately connected to the anesthesia machine to permit complete control of pulmonary ventilation by the anesthesiologist (Fig. 3).

(4) The child is then transferred to the fluoroscopic room, which in our case is located at some distance from the bronchoscopic operating room. The child's endotracheal tube can remain connected to the anesthesia machine at all times. The wheeled cart bearing the child can be rolled just ahead of a small anesthesia machine. Such transfer is not as convenient as doing the entire procedure on a fluoroscopic table but is necessary in our hospital, and the trip has been negotiated safely many times. Before the trip to the fluoroscopic room is started an electric suction machine is set up and tested there. A wheeled table containing the bronchoscopic instruments is also transferred to a point just outside the fluoroscopic room. All this makes quite a procession in the hospital corridor but it all seems necessary to the safety of the patient.

(5) After the child is in place on the fluoroscopic table, and the anesthesia is under proper control, introduction of contrast

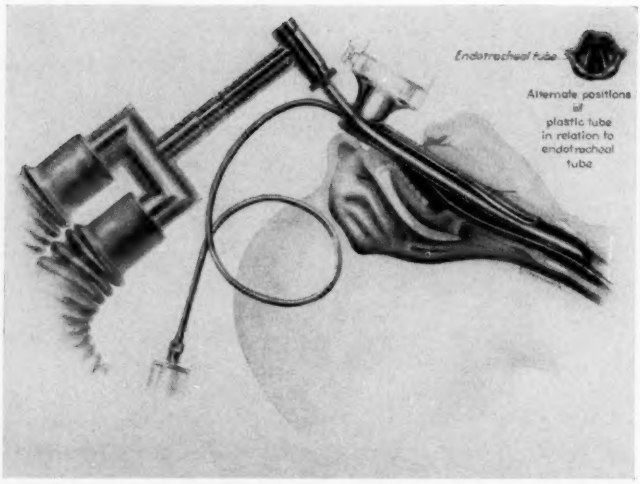

Fig. 3.—The plastic tube for injection of contrast medium lies in place as explained in Fig. 2. The anesthesia catheter has been introduced through the larynx alongside it and is connected to the anesthetic gas machine. The relative positions of the tubes in the larynx is shown in the inset. The syringe for injection of contrast medium is connected to the tube by a blunted 13 gauge needle.

medium is begun through the plastic tube while the child breathes through the endotracheal anesthesia catheter connected to the anesthesia machine. Even in the darkened fluoroscopic room the anesthesiologist always has complete control of respiration by constant palpation and compression of the rebreathing bag of the anesthesia machine. When roentgenograph is to be made the anesthesiologist "holds the patient's breath" by compression of the bag; he allows the breathing to start again spontaneously or breathes manually for the patient after the picture has been made. The patient's position is changed as desired to direct the contrast medium into various bronchi. Systematic coating of bronchi by the contrast medium is carried out under fluoroscopic guidance of the radiologist. He inventories the branch bronchi and is not satisfied until all have been demonstrated, or until adequate attempts at demonstration have failed.

Teamwork is needed to make children's bronchograms. Anesthesia is maintained by the anesthesiologist. Proper coating of the bronchi, with contrast medium, the making of "spot films," and

positioning of the patient to permit this is done by the radiologist and the personnel of his department. Injection of contrast medium and positioning of the tube through which it flows is done by the bronchologist.

Basal anesthesia with amylene hydrate (avertin) is usually employed. This anesthesia is reinforced by topical application of 2 to 4 percent cocaine in aqueous solution to the tracheobronchial tree after aspirating endobronchial secretions through the bronchoscope. Anesthesia may be further reinforced by intravenous injection of pentothal or pentothal-curare solution. Great care in dosage of anesthetic agents is taken. Adequate precautions to prevent accidental administration of intravenous medication in the darkened fluoroscopic room are used. The author is well aware of the criticism of pentothal-curare anesthesia for bronchography and relies on the excellent anesthesiologist with whom he works. It has seemed that with careful administration of anesthesia plus proper protection of the airway that this confidence has been justified.

Introduction of the bronchoscope after avertin has been given makes the patient cough and squeeze his bronchial secretions into the large bronchi. They are removed from these bronchi by aspiration. This clears the airway for adequate ventilation, and permits topical anesthetic agents to reach the bronchial lining and be effective.

After bronchograms have been made contrast medium is sucked out by a catheter through the endotracheal anesthesia tube or through the bronchoscope, and the child is observed closely in the post anesthesia recovery room until he can be sent to his own room with safety.

SUMMARY

A method for the making of children's bronchograms is described. It seems to satisfy the safety requirements by protecting the airway at all times, and allowing the operator control of the injection of the contrast medium with no embarrassment of respiration.

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LVIII

PRIMARY SPLIT THICKNESS SKIN GRAFTING OF RADICAL MASTOID CAVITIES

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Extended post-operative otorrhea from radical mastoidectomy wounds has long been a troublesome problem to those doing temporal bone surgery. The practice of skin grafting to obviate this difficulty is not new as indicated by the numerous references in the literature. Our small group of 26 mastoidectomy cases has given encouraging results with the immediate application of a split thickness skin graft utilizing a mold. We do not use this technique in our fenestration cavities. Since the tympanomeatal flap covers part of the cavity, Farrior's method of skin grafting these is more suitable.

A variety of cases make up the mastoidectomy group. Except for a few cases of cholesteatoma, these patients had uncomplicated chronic mastoiditis. At least three presented complications: preoperative facial paralysis, Bezold's abscess, and atresia of the aural canal with continuing foul discharge following simple mastoidectomy. Both postauricular and endaural approaches were used. The dental burr was employed for bone dissection.

Mosher[1] reported in 1925 on his "basket mold," in use since 1915. He employed paraffin gauze with a small amount of melted paraffin. This was used to make the mold for the application of his primary split-thickness graft in the radical mastoidectomy cavity. In five to ten days the gauze packing was removed from the basket, the basket walls collapsed, and the mold removed in toto through the canal. The donor site was dressed with paraffin gauze for five to ten days.

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Daggett and Bateman[2] of London, use the Thiersch graft through the meatus fourteen days after the radical operation. They use collodion and methylene blue on the donor site to keep the graft from rolling up after cutting. The dye identifies more readily the side of the skin which is to be applied to the mastoid wound. They apply small pieces through the meatus to line the cavity. A special wax preparation (Benzyl-naphthol, oil of eucalyptus, olive oil, paraffin) is poured into the cavity to form the mold and hold the graft in place. This wax plug is removed in two weeks.

Shambaugh, Jr.[3] used the Mosher "basket mold" and stated: "The use of primary skin graft hastens complete epithelization and prevents troublesome granulations, thus favoring preservation of the hearing." He reported success in modified radical mastoidectomies.

Boies[4] favors delayed grafting at the tenth to fourteenth postoperative day. It is his belief that "skin grafting shortens the period of epithelization sometimes by weeks."

Cunning[5] employs the Daggett-Bateman technique but puts the graft through the open wound instead of through the meatus.

Ballenger[6] outlines the above-mentioned procedures as well as the "crepe de lisle" method of Leon White.[7] A large skin graft is placed on crepe de lisle and inserted through the post aural incision. Iodoform tape is then packed into the cavity to fix the graft in place. The loose end is brought out through the meatus and the postauricular wound closed. White reported the end results in 100 cases of radical mastoid operations. His conclusion (before the days of chemotherapy and antibiotics) is that whether a primary, a secondary or no graft is used the outcome is about the same: approximately 50% good cavities. He seemed, however, to favor the Mosher paraffin basket.

Kopetzky[8] used the skin graft as did Mosher even when a plastic procedure was done on the posterior membranous canal wall. For holding the graft in place, he employed either a paraffin gauze packing, a paraffin tent, or a rubber finger cot "which was inserted into the tympanic cavity and then inflated and tied off."

More recently (1949) Farrior,[9, 10] has outlined his technique for primary skin grafting of the endaural mastoidectomy cavity. He uses it routinely both for mastoidectomy and fenestration. His procedure differs from those discussed in that surgical rayon is used as a dressing for the donor site and the cavity. Three separate strips of skin, backed by the surgical rayon, are inserted to line the various surfaces. Farrior[9] states: "If the cavity has been well lined with

skin, epithelization will be nearly completed at the end of the fourth post-operative week. Thereafter, one may adopt dusting powder, gentian violet, or drops until healing is complete."

Having briefly noted the prevailing ideas on this subject, we present now the technique which we advocate. Essentially, the same procedure was instituted in each of the 26 cases. Departures from it were minor and only made when especially indicated. In the beginning it should be said that we presuppose a clean mastoidectomy technique. Obviously skin grafting avails little in a poorly done mastoidectomy. Whether the approach was post-auricular or endaural, the dental burr was used, with magnification. Special emphasis was given the processus cochleariformis, the tensor tympani muscle and the tendon. The bony vertical facial ridge was lowered as far as the anatomical conformation allowed. In short, every effort was made to have a flat-walled, clean cavity without "overhang" before grafting.

For packing and making the mold, we have found a paraffin-vaseline impregnated gauze and rayon most satisfactory. A 4" x 4" piece of rayon is prepared as follows: equal parts of paraffin and vaseline are melted separately and then mixed together in a basin. In this mixture the 4" x 4" piece of rayon is place. After it is saturated with the hot mixture, it is lifted out with forceps and placed on a towel. The top of the towel is folded over and the excess of the mixture is removed. The piece is transferred to a container with a cover and autoclaved. It is kept in this sterile container until used. The half-inch paraffin-vaseline "Nu-gauze" is prepared in a similar manner except that the mixture is made up of 40% paraffin and 60% vaseline. The "Nu-gauze" is replaced into the original bottle for sterilizing.

The anterior aspect of the thigh is chosen as the donor site for obtaining the skin graft in these cases. The site is shaved, cleaned, and wrapped with sterile towels prior to the patient's arrival in surgery. The graft is usually taken by an assistant while the operator completes final preparation of the cavity. The assistant changes gloves prior to taking the graft to prevent cross-contamination. The site is re-prepared by scrubbing with soap and water, alcohol and ether and then painted with a 2% solution of methylene blue. While the skin is held tight with either suction cups, a flat retractor with teeth or sterile tongue blades, a split-thickness skin graft, paper thin, is taken with a Blair knife. The size of the graft depends on the size of the cavity to be covered. We usually cut a piece of skin approximately 2" x 3.5" so as to have ample available. After

the graft is taken, the donor site is covered with rayon and a pressure dressing of sterile waste and an "Ace" bandage is applied. This dressing is left undisturbed for two to three weeks.

Placement of the graft is facilitated by the following technique: The piece of rayon is placed in the cavity and the "Nu-gauze" is carefully packed into the depression and all the various angles of the cavity are completely filled. This combined gauze and rayon mass makes a cast of the cavity and, because of the paraffin impregnation, holds its shape when removed. The whole mass is removed by a steady outward pull. It must not be rocked from side to side unduly, or the shape will be lost. After delivery of the cast, the split thickness graft, large enough to line all parts of the cavity, is draped over the cast, methylene blue surface innermost (toward the cast). Using 0000 plain catgut with an incorporated needle, sutures are taken where the skin graft edges meet. The cast with the "gloved" skin is reinserted into the cavity. The free edges of the remaining rayon are cut off. The protruding edges of the graft are draped to cover the outer wall of the radical cavity. If the surgical approach has been post-auricular, the external canal wall plastic procedure is done at this point and the flap or flaps are tucked posterior (deep) to the Thiersch graft. The end of the half-inch "Nu-gauze" is brought out of the external canal opening. The incision, when post-auricular, is now closed. If the graft covers part of the skin of the external canal, no harm is done. In fact, a little excess skin is desirable to insure complete coverage of the epitympanum, tegmen and temporal muscle. These points of junction of the graft skin and attached skin are the most likely to form granulations later, unless precaution is taken to leave no raw surface. The graft over the attached skin dies and comes away post-operatively. The packing is left in the cavity for ten to twelve days. The half-inch strip comes out easily through the canal, followed by the remains of the rayon. In about ten to fifteen days the outer layers of the graft come away and give the impression that the entire graft is lost. But on more careful inspection a thin layer of new skin will be seen lining the healed cavity.

Post-operative care consists in doing as little as possible to the cavity. Sterile technique should be used in examination and treatment for the first three weeks.

Before turning to results, a definition of "healed cavity" or "dry ear" is in order. For our purposes, these terms mean a cavity that has epithelized, leaving no discernible granulation tissue or purulent exudate. The patient is not conscious of drainage from the ear. We consider such a one a "healed cavity."

A discussion of our results would mean very little without examination of a control group of cases. There are twenty cases in the control group. Radical mastoidectomy was performed with one of us on the case. They differ from the other group in that no skin grafting was done. In the control group, the shortest time required for a dry ear was six weeks. Two patients still have draining ears at five and ten years respectively, after operation. These two have refused re-operation. Shambaugh, Jr. mentions one of his patients who had a modified radical mastoidectomy without skin graft, whose ear drained for two and one-half years. The average time required for a healed dry cavity in the remaining group of 18 control cases is 25.6 weeks.

There were 26 cases in which the split-thickness skin graft was applied to the radical mastoidectomy cavity. Of this group, three ears are not included as dry. One graft did not take and the ear still drains at 19 months. Another in which a post-auricular incision was used had to be re-operated upon because stenosis of the auditory canal developed. The third "wet" ear is in a patient who shows poor healing ability as evidenced by marked delay also in the healing of the donor site on the leg. The remaining 23 patients had dry ears. Two recovered in as short a time as two and one-half to three weeks, and two in four weeks. The average time required for a dry ear in this group is 9.3 as opposed to 25.6 weeks in the control group. As previously stated, nothing is proven statistically by the observation of a group of this size. However, the order of difference between 9.3 weeks for grafted cases and 25.6 weeks for the control group, is notable. Further it indicates the value of immediate Thiersch grafting of radical cavities and tends to corroborate the opinions of Boies, Shambaugh, Jr., and Farrior as to the value of grafting.

COMMENT

No attempt is made to draw conclusions, however, we have learned certain facts.

1. A thin graft "takes" more readily in a mastoid cavity than a thicker graft, approaching full thickness. One adult had a Bezold's abscess complicating chronic mastoiditis. A thin split-thickness graft was applied to the cavity. Post-operatively the post-auricular wound broke down and there was a sizeable slough near the tip. In spite of this fact, however, the skin graft being thin took completely. In other cases it was noted that where the graft was thickest the healing was slowest.

2. An exposed facial nerve did not seem to interfere with the immediate graft. One patient had a pre-operative facial paralysis

complicating chronic mastoiditis. At operation a dehiscence was found in the intratympanic course of the facial nerve, near the horizontal semicircular canal. After the granulations were cleared away and the radical mastoidectomy completed, a thin graft was applied. Six weeks later the cavity was healed and the peripheral facial paralysis had cleared. The above experience is contrary to Ballenger's dictum: "The primary graft should not be used in an exposed facial nerve. . . . Exposed dura or lateral sinus are not contraindications for the graft." Boies takes the view that one should avoid grafting over exposed lateral sinus or dura. We found that dural, lateral sinus, or facial nerve exposures did not seem to impair graft healing. Since there is some difference of opinion, we feel that immediate grafting in all cases is worth a trial.

3. The donor site on the thigh is covered in seven-15 days with thin epithelium. The area of new skin will sometimes show bleb formation from the friction of clothing. This is especially true in the male patients because of friction from the trouser leg. In such cases the wounds require covering for a little longer period. As soon as the new skin has become thicker, it withstands friction.

4. The following have been discarded as unsatisfactory.

a. A dental or Stent mold, or other semi-rigid material, is impractical.

b. "Papering" the cavity with strips of skin only, does not work as well as draping and sewing over a removable mold. It is difficult to get the strips in place and even more difficult to get them to stay in place after the operation.

5. Skin grafting did not aid in conservation of hearing. The final loss for the operated ear was approximately the same in grafted and non-grafted cases.

SUMMARY

1. With 20 non-grafted cases as control, results of 26 cases of radical mastoidectomy, in which primary Thiersch skin graft was used, are presented. Included are cases of uncomplicated chronic mastoiditis and cases involving pre-operative facial paralysis, Bezold's abscess and atresia of the canal. Approaches were both endaural and post-auricular utilizing a dental burr and magnification.

2. A review of available literature is included.

3. Technique for taking the graft, placement of the graft using a vaseline-paraffin gauze and rayon mold for "gloving" the skin, and post-operative care are discussed.

4. Results, lacking sufficient cases for statistical proof, suggest the value of the procedure: epithelized, dry cavities in an average of 9.3 weeks in grafted cases as opposed to 25.6 weeks in non-grafted cases treated by the same operators.

5. Important to the interested reader are facts learned by trial and error: a thin graft takes best; exposed facial nerve, lateral sinus, or dura does not contraindicate grafting; the donor site on the thigh will sometimes blister (thin new epithelium) from clothing unless kept covered for a few weeks; there is no difference in conservation of hearing between grafted and non-grafted cases. Discarded as useless were Stent compound molds, and "papering" the cavity with strips of skin.

We express our gratitude to Doctor Herbert H. Harris, Professor of Otolaryngology, Baylor Medical College, for his co-operation.

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LIX

AN ENDOTRACHEAL METALLIC PROSTHESIS IN THE TREATMENT OF STENOSIS OF THE UPPER TRACHEA

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In the treatment of a case of chronic cicatricial stenosis of the upper trachea the feasibility of implanting a hollow cylinder of an inert metal in the lumen of the trachea was investigated. The ideal was a thin-walled, rigid, hollow cylinder, which could be placed within a stenotic segment of the trachea, after its lumen had been opened, and left for a prolonged period of time, to act as an airway and as a splint, around which a full-lumened trachea could reform.

A metallic cylinder, open at both ends has been chosen. It is made of an alloy of cobalt and chromium, an extremely hard and resilient material, highly resistant to chemical action. It is not malleable and therefore must be cast in the desired form. The finished cylinder is a very smooth, highly glistening metallic object. The alloy goes by the name of "Nobilium" and resembles the widely used alloy "Vitallium." Experience demonstrated that the use of this tube within the trachea was practical and feasible, but did not completely attain the ideal which had been hoped for.

While the cylinder did, indeed, act as an effective airway and splint, it caused interference with the highly important ciliary mechanism by which the tracheo-bronchial tree rids itself of mucus and foreign matter. Microscopic sections showed a loss of cilia in the portion of the tracheal wall around the cylinder and even a loss of the superficial layers of the epithelium in this area. There was an accumulation of mucus within the lumen of the cylinder; such mucus became inspissated and tended to act as an obstruction. Furthermore, the walls of the trachea surrounding the cylinder showed an inflammatory reaction in the animals studied: there was edema, vascular engorgement and leukocytic infiltration of the tunica propria. In the patient studied there was edema of the mucosa in the area directly above and below the margins of the cylinder.

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To the credit of this tube it may be said that, in spite of the disadvantages, it did serve as an effective prosthesis, providing a rigid splint for the trachea and serving as an adequate airway. The metal itself did not show any evidence of corrosion from prolonged contact with body fluids. It is felt that such a cylinder can be used for this purpose if its limitations are borne in mind and the patient observed at rather frequent intervals for the possible accumulation of inspissated mucus within the lumen and for edema of the mucous membrane above and below the upper and lower rims of the tube.

We were presented in 1948 with a difficult case of subglottic stenosis. Experience with a previous case, shortly before the war, in which we had used the classical laryngostomy and graduated sizes of indwelling rubber molds had not been entirely satisfactory. In our experience the rubber had proven to be rather irritating to the laryngeal tissues; the mold was continually bathed in foul-smelling mucopurulent secretions. The present case possessed certain special features: the stenosis was definitely subglottic, the vocal cords were of normal appearance and both cords moved actively in abduction and adduction; the voice was good, although somewhat weak. Therefore, it seemed highly important to us that any therapeutic efforts must not damage the vocal cords or the crico-arytenoid articulations. Repeated bouginage in this case had accomplished nothing, and a tracheostomy with implantation of a skin graft had not been successful.

A hollow cylinder of a non-irritating metal was decided upon. Using a non-corrosive metal, as a prosthesis, buried in the tissues, is not new but it has only been in recent years that suitable metals have been developed.

Canfield[7] reported a case in which he had used a silver tube sixteen years previously; it had been placed in the larynx after thyrotomy with removal of a stricture and had been left in situ for eleven months without untoward effect. It had then been removed and she had remained well for sixteen years.

Masmonteil[10] in 1935 reported good results with stainless steel. Menegaux and Odiette[12] in the same year carried out extensive studies on the reactions of osseous tissue to several metals. They found that three types of stainless steel gave no reaction in the tissue. Campbell, Meisrowky and Hyde[5] carried out tests on the cytotoxicity of various metals and alloys, based on the rate of growth of fibroblasts in tissue culture. They compared gold, silver, ticonium casting alloy, ticonium wrought alloy, vitallium, stainless steel, vanadium and copper. Copper and vanadium were found to be highly toxic; the others were not.

Campbell, Meirowsky and Tompkins[6] found ticonium casting alloy to be toxic, probably due to its beryllium content; its toxicity was not manifested until after about six months' lodgment in the tissue. However, Strock[18] reported satisfactory results in wiring a fractured mandible, after an open reduction, with ticonium wire.

In 1937 Venable and Stuck[19-21] demonstrated the injurious effects of electrolysis occurring within the tissues when dissimilar metals or certain alloys were used. They pointed out, in particular, the harmful galvanic effect of plated appliances, alloys containing iron and alloys in which the component metallic elements are not adjacent to each other in the electromotive series. The idea of galvanic action was challenged by Bothe, Beaton and Davenport,[3] who reported that the electrical potential developed between dissimilar metals within the tissues was not closely correlated with the local tissue reaction, except in the case of the more soluble metals which were strongly positive in the electromotive series. Venable, Stuck and their colleagues demonstrated the corrosive action (chemical, not electrolytic) of the body fluids on one single metal, when used as an appliance. They found the alloy vitallium to be almost perfect for use in the tissues. This alloy has been used extensively in almost every portion of the body.

Vitallium is an alloy of cobalt, 65%, chromium, 30%, and molybdenum, 5%; it is inert when in the tissues; it is not malleable, but must be cast into its final form. Pearse[15, 16] was the first to use a hollow tube of vitallium in one of the body cavities, namely in three cases of benign stricture of the bile duct, in two of which there was a successful outcome. Bettman and Tannenbaum[1] in 1945 reported a case in which a vitallium tube used to repair the common bile duct became plugged with a deposit. Such occurrences were also reported by Pearse[17] in 1946 and by Neibling and Walters[14] in 1947.

Other structures include the ureters (Lord and his colleagues[9]) arterial defects (Blakemore, Lord and their co-workers[2]) and the nasolacrimal duct (Muldoon[13]).

Another metal which has found wide application for implantation within the body has been tantalum. Tantalum is an element, discovered in Sweden in 1802 by Ekeberg. It is inert, malleable, ductile and can be procured in the form of suture material, ribbon, foil, plates and screws. The use of tantalum was first reported by Burke[4] in 1940; this metal soon found wide application, first in neurosurgery, then in all types of surgery.

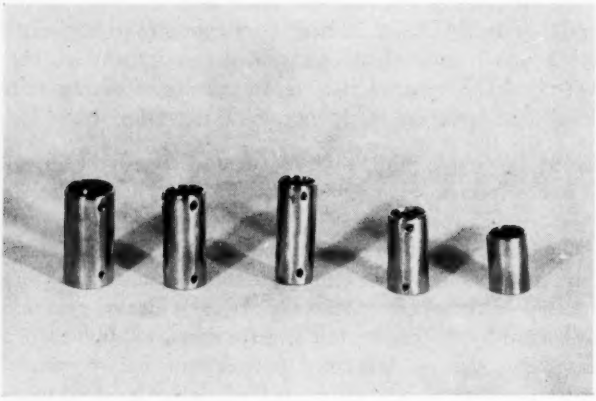

Fig. 1.—Nobilium cylinders, used in these observations.

EXPERIMENTS

It was decided to determine the tolerance of the trachea for an inert metallic cylinder on a series of animals. Preliminary trials with tantalum did not prove satisfactory. We had used a rectangular sheet of tantalum 0.007 inch in thickness, rolled into a cylinder. This improvised cylinder presented two disadvantages: first, its tendency to spring open; second, the sharpness of its edges, which we were unable to file down smoothly.

After this we turned to an alloy called "Nobilium." Nobilium is a chromium-cobalt alloy, used for casting full and partial dentures. Its physical properties are listed below, as supplied by the manufacturer:

Ultimate tensile strength	104,600 pounds/square inch
Yield strength	67,000 pounds/square inch
Elongation	4 per cent
Modulus of elasticity	24,000,000 pounds/square inch
Knoop hardness number	361

Chemical composition:

Cobalt	66.73 per cent, by weight
Chromium	27.29 per cent, by weight
Iron	0.05 per cent, by weight
Manganese	0.36 per cent, by weight
Silicon	0.22 per cent, by weight
Aluminum	0.01 per cent, by weight

We had a number of these tubes made of several sizes (Fig. 1). Some were two, others three centimeters in length. The outside diameters were 11, 12, 13 and 14 mm respectively. The wall thickness of all was one half millimeter. Either two or four eyelets were made near each end for the purpose of passing suture material through. The tubes were extremely hard, smooth surfaced, and had a high polish. The edges were nicely rounded and smooth, a feature which we considered to be desirable.

Seven dogs were used in these trials, the tubes being retained in the trachea, imediately below the larynx from two to eighty-five days. In all seven animals the same operative procedure was used. Nembutal was administered intravenously for anesthesia. The neck was opened through a vertical midline incision. The anterior wall of the trachea was exposed and incised vertically in the midline from just below the first tracheal ring, caudally for a distance of approximately three centimeters. The trachea was laid open and the cylinder was placed within the lumen and sutured to the tracheal wall by means of four stainless steel wire sutures, two at each end, passing through the tracheal wall. A longitudinal sliver of tracheal wall was trimmed off along the margin of the incision, in order to bring the size of the trachea down to make a snug fit around the cylinder. The tracheal incision was closed by means of interrupted sutures of No. 0 plain catgut. The pretracheal fascia was closed by means of mattress sutures of the same material. The ribbon muscles were overlapped and closed across the incision with mattress sutures of No. 0 plain catgut. The skin incision was closed with No. 1 black silk.

Dog No. 101 *C:* The animal apparently had no difficulty in breathing through the tube following the operation, however he refused all nourishment after the first twenty-four hours. At the end of forty-eight hours the animal was killed and the larynx and upper trachea removed. There was moderate brawny edema of the neck tissues in the operated area. The larynx and upper trachea were opened and the nobilium cylinder removed (Fig. 2, 101). A thin film of tenacious, inspissated mucus was noted within the lumen of the tube, coating the walls, but not of sufficient thickness to compromise the airway. On gross inspection the tracheal walls at the site of the tube appeared to be normal. Microscopic examination of a section of tracheal wall revealed a loss of the superficial layers of tracheal epithelium and loss of cilia. The tunica propria showed mild edema and engorgement of the blood vessels, with slight leukocytic infiltration of plasma cells and lymphocytes. There was some

Fig. 2.—Gross specimens, consisting of the tracheae laid open so as to expose the interior, from animals 101 C to 105 C, inclusive.

loss of substance, believed to have been produced during the process of sectioning.

Dog No. 102 *C:* The animal had no difficulty in breathing through the tube following the operation. He remained lively and ate well. After nine days he developed severe diarrhea (as did several other dogs in the same room) and expired on the tenth day. There had been no respiratory symptoms at any time. After death the larynx and upper trachea were removed; they were opened and the nobilium cylinder taken out (Figure 2, 102). A coating of tenacious mucus was found lining the inner wall of the cylinder, although it was not sufficiently thick to obstruct the respirations. On gross inspection the tracheal walls at the site of the cylinder appeared to be normal. Microscopic examination of a section of the tracheal wall, at the site of the cylinder, revealed a loss of the superficial layers of the tracheal epithelium and loss of cilia. The tunica propria showed mild edema and engorgement of the blood vessels, with moderate infiltration of lymphocytes.

Dog No. 103 *C:* The animal had no difficulty in breathing through the cylinder following the operation. He was lively and ate well. However, after three days he developed a severe diarrhea (as did several other dogs in the same room) and expired on the fourth day. There had been no respiratory symptoms. After death the larynx and upper trachea were removed; they were opened and

the tube was taken out (Fig. 2, 103). Only a few shreds of dried mucus were found on the inner walls of the cylinder. On gross inspection the tracheal walls at the site of the tube appeared to be normal. Microscopic examination revealed a loss of the superficial layers of the tracheal epithelium and loss of cilia. The tunica propria showed mild edema only.

Dog No. 104 *C:* The animal had no difficulty in breathing through the tube following the operation. He was lively and ate well. On the twenty-second post-operative day the animal was killed, even though he was still in excellent condition. After death the larynx and upper trachea were removed and opened (Fig. 2, 104). The tube was removed. A thin film of inspissated mucus was present on the inner wall of the tube, which did not interfere with respiration. On gross inspection the tracheal wall at the site of the cylinder appeared to be normal. Microscopic examination of a section of the tracheal wall, at the site of the cylinder showed an intact epithelial layer and cilia (portions of the cilia were lost in the preparation of the sections). The tunica propria showed some engorgement of the blood vessels and slight leukocytic infiltration with plasma cells and lymphocytes.

Dog No. 105 *C:* The animal had no difficulty in breathing through the tube following the operation. He was lively and ate well. On the fifteenth post-operative day the animal was killed. After death the larynx and upper trachea were removed; they were opened and the tube was taken out (Fig. 2, 105). Only a few shreds of dried mucus were found on the inner wall of the tube. On gross inspection the tracheal wall at the site of the tube appeared to be normal. Microscopic examination showed an absence of cilia without any apparent loss of the epithelial layers. The tunica propria showed engorgement of the blood vessels and rather marked leukocytic infiltration, principally with polymorphonuclear neutrophiles, and, to a lesser extent, with plasma cells and lymphocytes.

Dog No. 106 *C:* The animal had no difficulty in breathing through the tube following operation. Activity and appetite were good. On the second post-operative day there was slight leakage of mucus from the lower end of the incision, but by the fifth day this had ceased; the entire wound healed promptly thereafter. On the forty-second post-operative day the animal was killed. After death the larynx and trachea were removed; they were opened and the tube was taken out (Fig. 3, 106). There was a small amount of rather dry sticky mucus on the inner wall of the tube. On gross inspection the tracheal wall at the site of the cylinder appeared to

Fig. 3.—Gross specimens, consisting of the tracheae laid open so as to expose the interior, from animals 106 C and 107 C.

be normal. Microscopic examination showed an absence of cilia and a change from the normal respiratory type of epithelium to a stratified layer; this epithelial layer was much thicker than normal. The tunica propria showed edema, marked engorgement of the blood vessels and leukocytic infiltration, principally with polymorphonuclear neutrophiles, and, to a lesser extent, with plasma cells and lymphocytes.

Dog No. 107 *C:* This dog had no difficulty in breathing through the tube following operation. Activity and appetite were good. The wound healed promptly. On the eighty-fifth postoperative day the animal was killed. After death the cervical portion of the trachea was removed; it was opened and the tube was taken out (Fig. 3, 107). There was a light coating of dried mucus on the inner wall of the tube. On gross inspection the tracheal wall at the site of the cylinder appeared to be normal. Microscopic examination showed an absence of cilia, but otherwise the epithelium had retained its pseudo-stratified columnar form. The tunica propria showed no noticeable edema, nor any unusual engorgement of the blood vessels. There was only a moderate degree of leukocytic infiltration: plasma cells and lymphocytes, with only an occasional polymorphonuclear neutrophile.

In none of these trials did the nobilium cylinders show any evidence of corrosion; all of them retained their smooth surfaces and high polish.

CLINICAL OBSERVATIONS

G. N., a white female, aged sixteen years, was admitted to the Firmin Desloge Hospital on the Ear-Nose-and-Throat Service on September 15, 1948, complaining of obstruction of the airway, weakness of the voice and an unsightly scar on the neck. The history

of her trouble dated back to an attack of diphtheria at the age of three years. At that time a tracheotomy had been performed. The tracheotomy tube had been worn for a long time (the length of time it was worn was not known to us). After the tube was finally removed, the tracheal fistula remained open; breathing took place through the natural airway and the fistula. In 1940 the fistulous tract had been dissected out and the opening closed. Following this procedure, the patient had undergone a series of dilatations of the larynx and upper trachea, through a laryngoscope, over a period of approximately one year. There is said to have been improvement under this regime, and the patient received no further attention until the summer of 1948. During July, 1948, two dilatations had been carried out by direct laryngoscopy, using the Chevalier Jackson metal bougie. On admission to the hospital on September 15, 1948, the physical examination revealed nothing of interest except for the larynx, trachea and the neck. The vocal cords and supraglottic portion of the larynx were essentially normal; both vocal cords moved actively in abduction and adduction, and the voice was good, although somewhat weak. However, approximately one centimeter below the vocal cords there was a tough, shelf-like band of dense scar tissue across the anterior half of the trachea. It was estimated that this band of scar tissue reduced the lumen of the trachea by about one half. This airway was adequate for quiet respiration, but caused dyspnoea on exertion. In addition there was an unsightly, depressed scar on the anterior aspect of the neck over the upper trachea, measuring about two by four centimeters; it was rhomboid, with its long axis lying vertically.

On September 22, 1948, under local anesthesia, the trachea was incised through a midline incision, from immediately below the cricoid cartilage caudally for a distance of about three centimeters. None of the scar tissue was excised at this time; it was simply spread open. An obturator consisting of the finger of a rubber glove, packed firmly with sea-sponge, and tied securely at its open end, was laid within the lumen of the trachea. The trachea would not close completely around the rubber obturator. In the small gap remaining anteriorly a Thiersch skin graft was placed, and the connective tissue anterior to the trachea closed over this with mattress sutures. A number five tracheal cannula was inserted at the lower end of the incision. The skin incision was closed above the cannula. At the end of twelve days the wound had become foul-smelling and there seemed to be considerable inflammatory reaction in the soft tissues surrounding it. Consequently, the wound was reopened at this time and the obturator removed. The skin graft had not survived. The tracheal cannula

was left in for ten more days, and then removed, and the wound allowed to close. By this time the inflammatory reaction of the soft tissues had subsided.

During October and November, 1948, dilatations were again carried out through the laryngoscope. However, we were not satisfied with the results, and it was at this time that the decision was made to try the implantation of the metal cylinder.

On December 15, 1948, the patient was operated upon. The neck was incised by a vertical midline incision over the upper trachea, going directly through the old scar. The trachea was exposed and opened; all scar tissue in the anterior wall was excised, from immediately below the cricoid cartilage caudally for a distance of approximately two centimeters. One of the nobilium cylinders was laid in the lumen here and sutured to the wall with four stainless steel wire strands, threaded through the eyelets of the cylinder. The tube used was two centimeters in length, had an outside diameter of eleven millimeters, and inside diameter of ten millimeters, and a wall thickness of one half millimeter. The soft tissues, here consisting of rather dense fibrous tissue, were closed across the tracheal dehiscence with No. 1 chromic catgut mattress sutures; the skin was closed with black silk sutures. A tracheotomy was performed, as low as it was possible to go in the neck and a number six tracheal cannula put in place. This was done as a precautionary measure, as we did not feel perfectly certain that the metal tube would prove adequate for respiration. After five days the tracheal cannula was removed and the opening allowed to close. The patient continued to breathe well through the newly placed metallic cylinder. She was discharged from the hospital on January 12, 1949.

On February 14, 1949, this patient was readmitted to hospital for the purpose of resecting the scar on the front of her neck. On February 23, 1949, this scar was completely excised by means of a long, transverse, elliptical incision, following as closely as possible the natural lines of tension of the skin of the neck. A second incision, parallel to the first, was made through the skin over the anterior chest wall, approximately six centimeters lower; the intervening bridge of skin was undermined and mobilized and slid upward to cover the neck wound. We were able to close both incisions, and a firm pressure dressing was applied. These incisions healed promptly and the patient was discharged from the hospital on March 8, 1949. At this time the respiration was excellent and the voice was strong. We felt very much encouraged with the case.

All went well until June 6, 1949, when the patient rather suddenly developed severe dyspnoea. She was rushed into the hospital and an emergency tracheotomy was performed about three centimeters below the previous incision, through a small transverse incision. Direct laryngoscopic examination, carried out later, showed redundant, soft, flabby, edematous mucosa below the vocal cords, which overhung the upper edge of the nobilium tube like a curtain. Metal tracheal bougies of the Chevalier Jackson type, numbers 21, 24 and 27, were passed through the glottic opening and through the tube at this site.

It seemed to us that the tube she was wearing was too short, too small in diameter and located slightly too low in the trachea. Consequently, on June 29, 1949, the neck was re-opened through the transverse scar which remained from the plastic repair of February 23, 1949. The trachea was exposed and was found to have healed by firm fibrous tissue around the tube. The anterior tracheal wall was opened by a vertical midline incision, the stainless steel wire sutures were cut and the metal tube removed. At this time soft, flabby, edematous mucosa was observed, not only above the upper edge of the tube, but likewise just below its lower margin. The tube itself was as bright and smooth surfaced as when it was first placed in the trachea six months earlier; there were a few shreds of dry mucus on its inner surface. It was observed on gross inspection that the tracheal mucosa against which the tube had been resting was smooth, free from ulceration, and of good healthy color; it showed no ill effects from the six months' contact with the metal cylinder.

The incision was carried up through the cricoid and the thyroid cartilages and, for the first time, the larynx was laid wide open. The vocal cords were of normal appearance. A new and larger nobilium cylinder, having an inside diameter of twelve millimeters, wall thickness of one half millimeter, outside diameter of thirteen millimeters, and a length of three centimeters, was put in place immediately beneath the vocal cords (that is, up, within the ring of the cricoid cartilage), and sutured in place by means of stainless steel wire strands, just as the first tube had been. The larynx and trachea were allowed to close, and the soft tissues directly anterior to the incision were closed with No. 1 chromic catgut mattress sutures. The tracheotomy tube which had been put in on the 6th of June was removed at the end of the operation, and this opening was closed.

Following this procedure the patient remained well; breathing was unobstructed and free. In September, 1949, a direct laryngo-

scopic examination was made, and a small amount of soft, flabby, edematous mucosa was noted in the subglottic region, immediately around the upper margin of the metal tube, although not as marked as in June, 1949. This tissue was punched out with a laryngeal punch forceps.

The patient remained well until December 15, 1949, when, during a hard spell of coughing, the tube broke loose and was aspirated into the right main stem bronchus. It did not seem possible to bring this large cylinder out through the trachea and glottis. Consequently, a low tracheotomy was again performed through the site of the emergency tracheotomy of June, 1949, using the same short transverse incision; a seven millimeter bronchoscope was passed through the tracheotomy opening, the cylinder grasped with forceps and extracted. The tracheotomy wound was immediately closed. This cylinder, which also had been in the patient's trachea for six months, remained glistening and smooth. A few shreds of dry mucus were noted on the inner surface of the cylinder.

The patient has remained well since that time. She was last seen in September, 1951, twenty-one months since the second tube broke loose and was removed. Laryngoscopic examination showed that the subglottic area appeared to be retaining its lumen; the anterior tracheal wall just below the glottis was very white and smooth, having the appearance of dense fibrous tissue, but covered with intact mucosa. She has had no further respiratory embarrassment and has a good voice.

CONCLUSIONS

A rigid, hollow, thin-walled cylinder of a cast, metallic, nonmalleable alloy, called nobilium, was implanted in the tracheas of a series of dogs for periods of time varying from two to eighty-five days, and in the trachea of one patient for two successive periods of six months as a means of treatment for a long-standing stenosis.

It was found that:

Such a cylinder, anchored within the trachea, usually resulted in a loss of cilia, and even a loss of the superficial layers of the tracheal epithelium in the portion of the tracheal wall immediately surrounding the tube, although this was not true in all instances.

There was always more or less of an accumulation of dry mucus within the lumen of the tube, which resulted in slight narrowing of the lumen.

Such a tube does provoke a limited amount of inflammatory reaction in the surrounding tracheal wall, as shown by the edema,

vascular engorgement and leukocytic infiltration of the tunica propria of the tracheal wall noted in the observation on animals, and the edema of the tracheal mucosa noted in the patient.

Such a tube, in spite of these reactions, does serve as an effective, rigid splint within the trachea, allowing the tracheal wall to heal satisfactorily around the tube, after obstructing scar tissue has been removed.

Such a tube serves as a satisfactory airway within the trachea, eliminating the necessity for a tracheal cannula.

The metallic alloy used for the tube did not show any gross evidence of chemical corrosion, even after a period of six months in contact with the tissues.

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LX

AGENESIS OF THE LUNG WITH TRACHEAL STENOSIS

CASE REPORT

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Agenesis of the lung like similar abnormalities which occur in early embryonic development is infrequent, and the diagnosis has not often been made during life. This diagnostic possibility must be kept in mind and with the increasing use of bronchoscopic and bronchographic observations in infants and young children can be more readily detected.

Most of the reported cases were stillborn or died in infancy from respiratory disease. Of the 87 cases recorded by Wexels[1] 28 were diagnosed during life; 13 of these were verified at autopsy, and when last heard of 15 were alive. Approximately a half of the total number of cases had other associated congenital defects such as accessory rudimentary ribs, atresia of larynx, imperforate anus, patent ductus, etc.

Unless complicated by other anomalies agenesis alone is not incompatible with a normal life span since one recorded case lived to the age of 72. Only one normally functioning lung is required to allow an individual to lead a nonrestricted life as evidenced by the increasing number of patients who, having undergone pneumonectomy, continue a normal existence. However, patients with one lung do not tolerate respiratory infections well, and when bronchopulmonary complications occur they become serious. Almost 50% of the recorded cases of agenesis of the lung died in the first five years of life generally from lung infection.

During the period of Germanic Classification at the turn of the last century Schneider[2] divided pulmonary agenesis in 3 classes:

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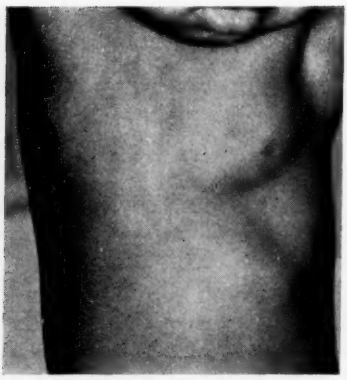

Fig. 1.—Photograph of patient. Flattening of left side of the thorax is evident.

(1) total absence of the lung, (2) rudimentary bronchus without lung tissue, and (3) bronchus with vestigial lung buds. The case herein reported falls into the second group.

Agenesis represents a true malformation, resulting from an inherent defect in the germ plasm. This abnormality has its origin when the embryo is in the 3-5 mm stage, for at that time the laryngotracheal groove which runs lengthwise in the floor of the gut has developed into a ridge with lung buds.

The symptoms consist of difficulty in breathing, cough and wheeze, although the condition may remain unrecognized during life because of the lack of symptoms. Without complications cyanosis is infrequent. Labored and wheezing respirations after birth suggest the possibility of a congenital anomaly, and agenesis of the lung may be responsible for these changes.

On physical examination the respiratory movements of the thorax are commonly not altered. Flattening of the affected hemithorax is the most frequently observed abnormality. Dullness on percussion with absent breath sounds and displacement of the heart, trachea and mediastinum to the affected side is found. These findings must be differentiated from pulmonary atelectasis, plugged bronchus, bronchiectasis, tumors, and tuberculous stricture.

The diagnosis is made by roentgenographic, bronchographic and bronchoscopic examinations. An angiocardiogram may reveal other anomalies of the cardiac and circulatory system.

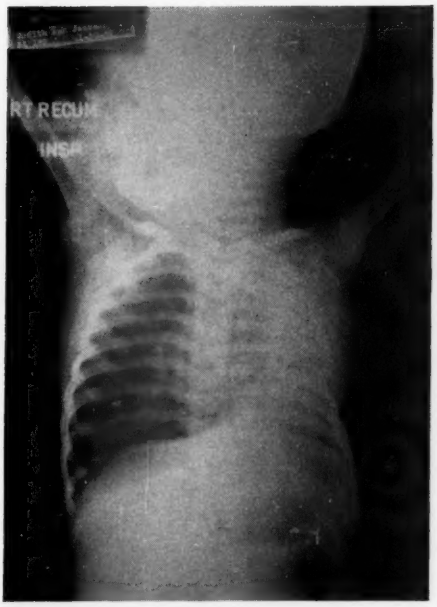

Fig. 2.—Rudimentary left 6th rib, asymmetry of the left thorax and absent aeration of the left lung with compensatory emphysema of the right lung as seen on the roentgenogram.

No aerated lung is visible on the roentgenogram, while the normal lung may protrude across the midline. Narrowing of the intercostal spaces, flattening of the costal contour, and an elevated diaphragm are usually noted on the affected side. Bronchoscopy and bronchogram reveal displacement of the trachea and the presence of a main bronchial bud or complete absence of a main bronchus with no tracheal bifurcation. Other abnormalities as stenosis or supernumerary tracheal rings are often disclosed.

CASE REPORT

J. J., one year old colored female child was admitted to Jefferson Hospital January 10, 1952, with the complaint of a croupy cough since birth associated with dyspnea and stridor. The child had practically resided in a hospital since the time of birth. In spite of the dyspnea the child was well-nourished and of normal structure.

Physical examination revealed flattening of the left chest (Fig. 1), loud, coarse rales throughout the lung, and moderate scoliosis

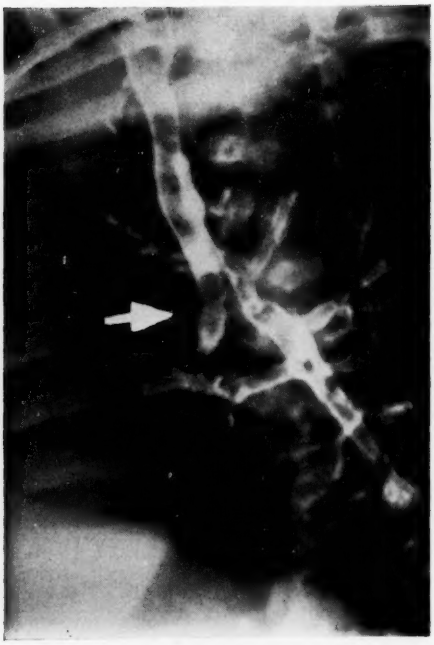

Fig. 3.—Bronchogram (oblique view) revealing a rudimentary left bronchus ending blindly.

to the left. Roentgen and fluoroscopic examinations disclosed no aeration of the left lung with a rudimentary 6th rib on the left and a compensatory emphysema of the right lung, suggesting aplasia of the left lung (Fig. 2).

On examination with a 4 mm bronchoscope the larynx appeared to be somewhat smaller than normal. The trachea was narrowed in its antero-posterior diameter, and because of this the bronchoscope could not be inserted more than 3-4 cm. No left bronchus could be observed. A bronchogram was done under general anesthesia, and this revealed a normal bifurcation of the trachea but with only a portion of the left main bronchus outlined, the remaining portions of the left bronchus being absent (Fig. 3).

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LXI

EFFECTS OF CORTISONE ON EXPERIMENTAL LYE BURN OF THE ESOPHAGUS

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Strictures of the esophagus following lye burns are fortunately less common than in the past. However, these cases, though less frequent, are still a serious problem. A severely burned child was seen in February, 1951. His condition was so acute it was evident that prolonged dilation therapy would be necessary to prevent cicatricial stenosis.[1-10]

The effect of cortisone on connective tissue has been well established. Granulation tissue and wound healing may be prevented by the use of cortisone. Howes[11] and Spain[12] demonstrated almost complete inhibition of inflammatory response and subsequent granulations following mechanical injury. Spain found that there was no difference in the granulations between treated and control rats, if the administration of cortisone is delayed 48 hours after injury.

Having this in mind, cortone was used as described in the case presentation, with very encouraging results. It was evident further investigation was needed. Dogs were used in order to obtain fair trial series in reasonable time. During the course of the experiment further encouragement was given by the work of Rosenberg, et al,[13] who found no stricture due to lye burns in rabbits treated with cortisone, but did note a greater number of deaths due to septic complications in the treated animals.

TECHNIQUE

The method used was that of Bosher, et al,[7] with certain modifications. The first two animals, No. 78 and 28, underwent gastrostomy. The lye was instilled while the cardia was manually clamped. It was found that the animals were able to take liquids within 24 hours and return to a soft well-balanced diet with four or five days.

The author acknowledges with thanks the kind assistance of Dr. Fred Kreutzer and Dr. Alan Johnson of the Department of Surgery, University of Calif. Medical School.

Fig. 1.—7-day cortone treated. Note inflammation and flat granulations.

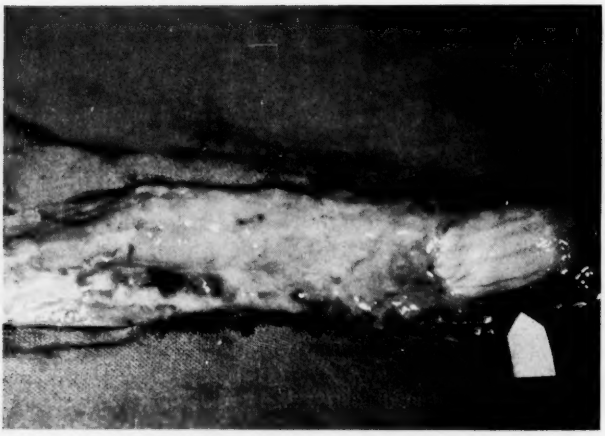

Fig. 2.—7-day control. Note large ulcer, exudation and heaped up granulations.

The remaining animals were treated without gastrostomy or endotracheal intubation in the following manner: After nembutal anesthesia the dog was placed in the Trendelenburg position. A levine tube was passed to the cardia. At first this was checked by esophagoscopy but it was found that the lower xiphoid margin was an excellent guide to the necessary length of tubing. A short length of tubing was connected to a suction machine and passed just through the cricopharyngeus. Tap water was then introduced into the Levine tube, carried by gravity up the esophagus, and withdrawn via the suction tube. This served to show that the system functioned. (In one case, No. 77, before using this precaution, the suction became obstructed, with subsequent overflow of lye into the trachea causing a large tracheo-esophageal fistula.) 20 cc of 10% lye solution was then instilled. After 60 seconds 40 cc of tap water was flushed through the system. The lye was not neutralized. The treated animals were immediately placed on cortone, 75 mgm per day in two doses given intramuscularly, and terramycin, 500 mgm per day orally. The terramycin was given prophylactically to control infection due to the loss of body response while under cortone treatment. The untreated animals were given no medication except as indicated in Table I.

Fifteen experimental animals were used. The animals died or were sacrificed over periods ranging from seven to 120 days. The results are tabulated in Tables I and II. There was a clear cut clinical difference between the control and treated animals except as noted. The untreated animals, even those that survived and did well, would continue to vomit profuse thick mucus for several weeks. The treated animals responded rapidly. At the end of the first week their behavior was no different from normal animals in the kennel.

GROSS FINDINGS

The control group all had marked esophageal granulations in the early period. If allowed to live over five weeks, all developed strictures. None of the strictures were complete. This corroborates the work of Bosher and his group.[7] The dog's esophagus may not react and close off completely as does the human. In fact, the stricture seems to be less tight at 120 days than at 35. The dogs' habit of bolting food may possibly act as a dilator. No. 128 was atypically normal from the beginning. He should be considered a technical error. Diluted lye may have been used.

No strictures were found in the treated group. The granulations, when present, were very much thinner and there was little

Fig. 3.—120-day control showing stenosis of mid-esophagus.

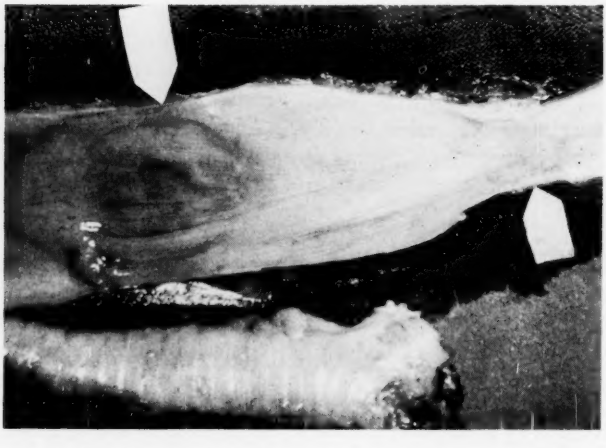

Fig. 4.—120-day control showing marked scarring. Healed ulcer.

inflammatory reaction. There were very few complications as described by Rosenberg, et al,[13] in the rabbits treated with cortone. In their series the mortality rate was twice as high due to perforations, pneumonia and mediastinitis in the treated animals. Their animals did not routinely receive an antibiotic. The rabbit response to cortone is apparently more active. In the Howes[14] series absolutely no granulations were found in the injured rabbit ears, whereas in our series the dogs did show a granulation reaction, though much less than normal. This also may explain the lack of perforations, mediastinitis, etc. in this series. A factor to be considered in future work is the affect of the terramycin. However, an antibiotic alone will not prevent stricture. Dogs No. 78 and 28 received penicillin intramuscularly for the first week. Both showed marked lye burn reaction as compared to the cortone treated animals.

MICROSCOPIC FINDINGS

Representative specimens were removed from all the animals' esophagi. Routine histopathologic sections were made and stained with hemotoxylin and eosin. There were several criteria helpful in determining the extent of the burn. All showed increased vascularity. The blood vessels had thick walls. The cells were almost cuboidal in many cases. With severe burns there were no mucous glands, the submucosa being filled with connective tissue. The most severe reactions showed loss of muscle.

The findings are similar to those reported by Bosher, et al.[7] Since the earliest specimen was seven days most of the slough that they described had already occurred.

The initial difference between the control and treated group during the first two weeks was in the amount and extent of exudation, which was markedly increased in the controls as compared to the treated animals. There was a tendency for deeper necrosis involving the muscularis and for greater loss of patent blood vessels in the early controls.

There is a constant progression of fibroplasia starting from the deeper granulations, thus tending to wall off the injured area. This is complete by the time epithelization has occurred. After six weeks there is less microscopic difference between the control and treated dogs than would be expected from the gross appearance. There is a gradual dissolution of the dense scar tissue seen at five weeks so that by ten weeks the submucosa is almost areolar, though more fibrotic than normal. This is true even in the sections taken from the stricture.

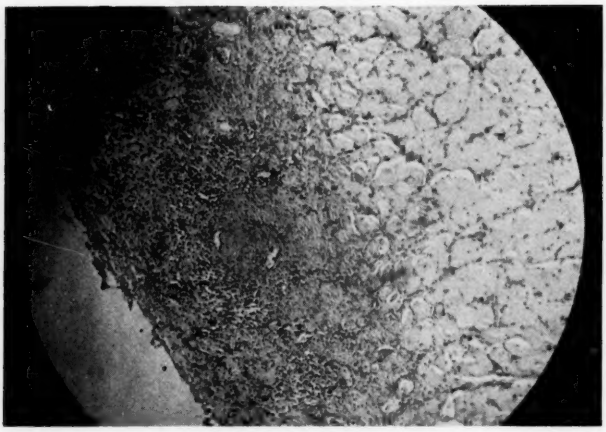

Fig. 5.—Microcsopic section of 35-day control. The entire submucosa and part of the muscularis is replaced by fibrous connective tissue.

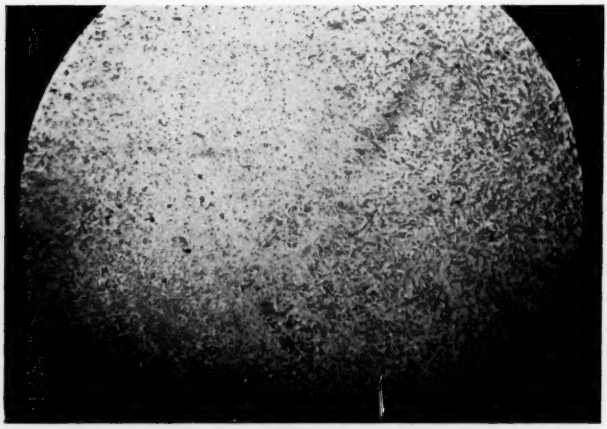

Fig. 6.—Photomicrograph. Seven day control. The granulation tissue is more dense and extends well into the muscularis, which is also involved.

SUMMARY AND DISCUSSION OF THE EXPERIMENTAL WORK

1. A method of consistently producing lye stricture of the dog's esophagus is described. There was one exception, which was probably a technical error.

2. Cortone was given over a six-week period. This is longer than necessary, but covers the entire period of healing and fibroplasia. The dosage—75 mgm per day—approximates in the experimental animal the amount that would be used in a child during the acute period.

3. Terramycin, 500 mgm per day—was used in conjunction with the cortone therapy as a protective measure against overwhelming sepsis.

4. The morbidity in the treated animals was decreased.

5. Cortone therapy diminished the local inflammatory reaction and granulations.

6. No stricture developed in the treated group, whereas all control animals living over 35 days developed a stricture.

7. The following questions have not been answered and deserve further study:

(a) Will cortone given immediately have a protective action in preventing deep necrosis into the muscularis? This necrosis may be a toxic reaction and not necessarily due to the direct action of the lye.

(b) Is it necessary to wait for "protective" granulations to develop in order to prevent perforations? The work of Spain, et al, would also speak against delay. A delay of 48 hours in administering cortone resulted in granulations comparable to the untreated rats.

(c) What role does control of infection have in the ultimate survival of the animals?

(d) Over what period of time should one give cortone to obtain optimum results?

REPORT OF CASE

S. H., a 17-month old boy, was seen in February, 1951, 30 minutes after drinking a concentrated lye solution. He showed the classical picture of a severe lye burn. His lips, tongue, palate and pharynx were swollen and red. There were scattered ulcerated patches. He was unable to swallow. The palate and uvula edema

was sufficient to cause concern as to the patency of the airway. Tracheotomy, although considered, was not necessary. A Levine tube was passed and the stomach contents evacuated. The tube was left in place. This offered adequate means of maintaining his fluid, food and medication. Cortone, 25 mgm, and terramycin, 250 mgm, every six hours was immediately started.

24 *hours:* The edema had subsided. Temp. 101° (R). The mucous membranes were now angry red and superficially ulcerated. There was slight hoarseness but no difficulty in breathing.

48 *hours:* The mucous membranes were normal except for superficial redness. The voice was normal.

72 *hours:* No evidence of reaction in the mucosa. Esophageal x-ray studies with radiopaque were normal. Cortone was decreased to 25 mgm q12h and terramycin 250 mgm q8h. The patient was started on oral feedings, which were well tolerated.

5th day: Esophagoscopy revealed shallow ulcers covered with a gray membrane. These were circumferential at the cricopharyngeus at the crossing of the left bronchus and at the cardia. There were numerous scattered patches on the posterior wall. There were no granulations or edema.

6th day: Discharged. Readmitted in the evening with temperature of 104² (R). All findings, including chest x-ray, were normal. He was placed on penicillin. The cortone and terramycin were continued. The patient was afebrile in 24 hours.

8th days Discharged. Cortone therapy, 25 mgm per day, was continued until the 12th day. It was discontinued only because the available supply was exhausted.

Esophagoscopy was repeated one month after the accident. The findings were normal. The most recent x-ray studies of the esophagus were done in March, 1952. They were reported as being normal. The child has had no difficulty. No dilatation was done since he was under close observation and there was no clinical or roentgen evidence of stricture or other complications.

CONCLUSIONS

1. The experimental evidence presented shows the advantage of cortone therapy in preventing stricture of lye burns in the dog's esophagus.

2. The morbidity of the treated animals was discussed.

3. In the experimental animal cortone therapy diminished the local inflammatory reaction and granulations.

4. Antibiotic therapy is an essential in the control of secondary infection.

5. All control animals living over 35 days had stricture formation. No strictures were found in the treated group.

The author wishes to thank Dr. Lewis F. Morrison, Chairman of the Department of Otolaryngology, U. of Calif. Medical School, for his kind assistance in making this work possible.

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TABLE I.—CONTROLS.

DOG NO.	DAYS OF EXPERIMENT	CLINICAL CONDITION	CAUSE OF DEATH	GROSS FINDINGS
*78	7	Poor. Much dribbling of mucus. Ate poorly. Penicillin. Post op. 7 days.	Killed	Inflamed markedly. Thick granulation. perioesophagitis.
168	7	Did poorly. Emaciated.	Killed in dog fight	Marked granulations.
180	16	Extreme dehydration with emaciation (5-mo. old pup).	Killed	Patchy granulations. Atrophic in spots.
108	19	Emaciated, eats poorly. Vomiting, malnutrition.	Killed	Thick red granulations. Narrowed lumen.
*28	35	Poorly 1st week, then apparently normal. Vomiting in 5th week. Penicillin. Post op. 7 days.	Killed	Strictures mid esophagus. Thin ulcer above stricture, adhesive to trachea.
15	47	Very sick for 3 weeks. Given terramycin 500 mg/day for 1 wk. with improvement.	Killed	Stricture of middle 1/3 esophagus.
128	98	Did well from beginning.	Killed	Normal appearing esophagus. ? technical error.
1	120	Vomiting 6 weeks. Gained thereafter.	Killed	Stricture of middle 1/3. Dilated above.

*Gastrostomy—fed via tube 4 days.

TABLE II.—CORTONE/TERRAMYCIN TREATED ANIMALS.

DOG NO.	DAYS OF EXPERIMENT	CLINICAL CONDITION	CAUSE OF DEATH	GROSS FINDINGS—ESOPHAGUS
14	7	Difficulty in eating for 3 days, then apparently normal.	Killed	Normal thickness. Thin erosions & granulations.
77	7	Very poor — emaciated. Constant coughing and vomiting. Technical error. Lye spilled into trachea.	Killed	Large clear esophageal—tracheal fistula. No granulations .
107	19	Ate well. No vomiting after initial period.	Killed	Slight injection. Few superficial erosions. No granulations.
149	20	Did poorly. Symptoms of distemper.	Died Pneumonia	Bile stained. Flat granulations.
177	70	Normal after initial period.	Killed	Normal appearance.
101	91	Normal after initial period. Delivered normal litter after 4 weeks.	Killed	Normal appearance.
94	120	Normal after initial period.	Killed	Normal appearance.

The Scientific Papers of the American Otological Society

LXII

ANATOMICAL CONSIDERATIONS OF MÉNIÈRE'S DISEASE

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The last decade in otology has been marked by a renewed interest in Ménière's disease and significant discoveries in pathological changes and therapeutic management. The symptomatology of this entity seems to have occasioned little interest, it being taken for granted that its clinical manifestations were those of a symptom complex of typical and well-known features.

From time to time, however, unusual phenomena in the behaviour of this condition have been described and recorded. The authenticity of some of them should be questioned. In so doing, some little reward may be found in a critical study of the anatomy of Ménière's disease and a recounting of symptoms which properly belong to this entity. By the same token, we shall find reason to question the relationship between Ménière's disease and those symptoms with which anatomical considerations are incongruous.

When reference is made to Ménière's disease, it is assumed that we are dealing with a lesion in the internal ear, a pathological change in the end-organ of the auditory nerve, which produces a well-defined symptomatology. A simple glossary will reiterate the clinical signs and symptoms, namely an attack of vertigo, nausea and vomiting in the severe seizures, a spontaneous nystagmus of varying degrees, a perceptive type of hearing impairment, sometimes bilateral but frequently more profound in the ear to which the vertigo is referred, and a tinnitus which is frequently aggravated immediately preceding and during the attack. The vestibular tests are often of little sig-

nificance. In one patient we may observe hypo-active responses. In another, a hyper-active labyrinth seems to be in evidence, while in other individuals there are no significant changes in the vestibular responses which truly identify the lesion responsible for the symptom complex.

Thus, we may list the signs and symptoms of Ménière's disease in a few brief words. They represent the absolute and complete clinical picture. They tell the whole story. Any stimulus that originates within the internal ear and is discharged along the auditory nerve to the vestibular nuclei in the pons and medulla must necessarily produce symptoms which are limited to this category. One may elaborate upon them by way of emphasizing that the hearing impairment might have long antedated the vertiginous attack, that it may be characterized by distorted sounds and fluctuations in degree, that the tinnitus is high or low pitched, that the vertigo is accompanied by falling and essentially that of the world turning about the patient, but in the last analysis, dizziness, nausea and vomiting, nystagmus, subjective noises and impaired auditory function complete the narrative.

You will note that I have enumerated these classical symptoms at the risk of pointing out the obvious. Why? Because there is often a tendency to spread our interest over a wider neurological field and to ascribe concomitant or associated neurological findings in isolated instances to the symptomatology of Ménière's disease. This only tends to confuse the issue and revive the acrimonious dispute recorded in otological history in regard to what constitutes Ménière's disease, Ménière's symptom complex, and Ménière's syndrome.

To be specific, your attention is called to certain signs and symptoms which have been reported in the literature to be integral parts of Ménière's disease. But first, I should like to describe the anatomical pathways traversed by stimuli which originates within the vestibular labyrinth. They are carried into the brain from the ampullary ends of the semi-circular canals over the vestibular nerves with cells of origin in the vestibular ganglion, and end in the four vestibular nuclei (superior, medial, lateral and inferior) and in the cerebellum. The medial and lateral vestibular nuclei, are believed to be related particularly to impulses from the horizontal canals. From the medial and lateral vestibular nuclei, fibers cross the midline and

ascend in the medial, longitudinal fasciculus to the abducens nucleus where they synapse with motor neurons supplying the lateral rectus muscle and with parabducens cells. These latter cells send their neuraxes forward through the medial longitudinal fasciculus to the portions of the oculomotor nuclei supplying the contralateral medial rectus.

Irritative lesions of the lateral or the medial vestibular nuclei produce a horizontal nystagmus with the quick component toward the side of the irritation. Irritative lesions of the superior vestibular nuclei produce a vertical nystagmus by similar connections through the medial longitudinal fasciculus. Irritation of all the vestibular nuclei or the vestibular nerve may produce a rotatory or irregular nystagmus.

Intercolated neurons from the medial vestibular nucleus carry impulses to the dorsal efferent nucleus of X. This nucleus has preganglionic neurons distributed by way of the myenteric plexus (ganglia) in the walls of the digestive tract to the smooth muscles of these organs, including the stomach. Consequently, stimulations of the vestibular apparatus may set up a reverse peristalsis with nausea and vomiting. (Pain in the stomach is mediated over splanchnic nerves.)

Turning now to some of the unusual symptoms which have been attributed to Ménière's disease, we find that the literature refers to a burning sensation in the throat. The glossopharyngeal nerve carries visceral sensations from the back of the pharynx which is covered by an entodermal epithelium. The cells of origin of these neurons are in the petrosal ganglion on the glossopharyngeal nerve (Fig. 3, right or left). The termination of these fibers is in the nuclear gray along the fasciculus solitarius. Irritating lesions at the acusticoglossopharyngeal level could well produce paresthesias such as a burning sensation of the pharynx. This symptom, however, could not come from an uncomplicated Ménière's disease.

Again we read in the literature of another unusual symptom of Ménière's disease, namely, a numbness of the face and ear on the affected side. The term "dead face" has been applied to it. A study of the anatomical relationships of the nervous system demonstrates that the cutaneous innervation of the face is mediated over the following nerves: 1, The fifth nerve with cells of origin in the semilunar ganglion. 2, The seventh nerve with cells of origin in the geniculate

ganglion. 3, The ninth nerve with cells of origin in the superior petrosal ganglion, and 4, the tenth nerve with cells of origin in the jugular ganglion. To be more specific, the cutaneous innervation of the ear is over the seventh, ninth, and tenth nerves. On entrance to the brain, the fibers arising from the cells of these several ganglia (which are related functionally to carrying impulses set up by cutaneous stimuli) enter the spinal tract of V and descend in this pathway to end at the lowermost medulla and upper cord (1-4C) levels. Irritative lesions of this spinal tract of V might produce symptoms resembling a tic while destructive or blocking lesions would produce a numbness of the cheek and ear to pain and temperature, but with some remaining tactile sense.

It should be emphasized that if the peripheral part of the fifth nerve is affected by a lesion, one may observe a sensory loss of pain, temperature and tactile, as well, perhaps, as a disturbance in its motor function. If, however, the spinal tract of the fifth nerve is affected, one observes a loss of pain and temperature with preservation of the tactile sense. The latter symptomatology is to be expected, namely, a preservation of the sense of touch when one observes that the lesion in the spinal tract of the fifth nerve is below the nucleus which deals with tactile sense.

Thus, we observe once more that numbness of the face and ear or a typical tic douloureux cannot be explained on the basis of an uncomplicated Ménière's disease. These phenomena are reconcilable only with a large lesion at the base of the brain encroaching upon the spinal roots of five, seven, nine and ten, or more logically explained as a lesion within the pons and medulla affecting the spinal tract of the fifth nerve.

CONCLUSIONS

1. Impulses resulting from the end organ lesion in Ménière's disease travel along a definite anatomical route and therefore produce clear-cut, well defined signs and symptoms as enumerated in the above glossary. Any deviation from this symptomological category suggests the presence of pathological changes in other parts of the nervous system.

2. When vertigo dominates the clinical picture but is associated with a burning sensation in the pharynx, numbness on one side of the face and ear, or a typical tic douloureux, it is impossible

to reconcile these findings with an isolated lesion in the internal ear. Their presence is an indication for study of other regions of the central nervous system. Within the category of possible pathological changes are such lesions as acoustic neuromas, cerebello-pontine angle tumors, aneurysms, and vascular anomalies, multiple sclerosis, and other degenerative changes with the medulla and pons.

(Discussion appears on page 738)

LXIII

OBLITERATION OF THE ENDOLYMPHATIC SAC AND THE COCHLEAR AQUEDUCT

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In spite of much investigation there is little factual information regarding the fluid physiology of the inner ear. Attempts at chemical analysis, for example, have been only partially successful because of the minute quantities of these fluids. The relation of endolymph and perilymph to each other, in regard to osmotic pressure, protein and electrolyte content, has not been settled. Likewise the origin and site of absorption of the fluids is not entirely clear.

On the basis of anatomical studies the prevailing opinion holds that endolymph is secreted mainly by the stria vascularis. Many believe that it is resorbed by the endolymphatic sac and duct.

Because the perilymphatic space is connected with the subarachnoid space by the periotic duct there is good reason to believe that cerebrospinal and perilymphatic fluids are identical. It is reasonable to suspect this duct of providing some physiological function in relation to perilymph.

This paper will concern itself with two questions in the following order:

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Support for these experiments was provided in part by a grant from the Central Bureau of Reasearch of the American Otological Society, Inc., in part by Contract No. N6-ori-20 T.O. XXIV ONR Project NR 140-608, and in part by the University of Chicago.

1. Will destruction of the endolymphatic sac and duct result in demonstrable inner ear changes?

2. Will interruption of the perotic duct cause demonstrable inner ear pathology?

An answer in the negative would imply that these structures either have no important function or that their function can be assumed by other structures in their absence.

In either case such an answer would detract strongly from those theories which ascribe certain ear diseases (particularly Ménière's disease) to abnormal function of these structures.

PART I.—ENDOLYMPHATIC SAC AND COCHLEAR AQUEDUCT

Guild[1] in 1927 presented a detailed account of the anatomy of the endolymphatic sac and duct in the guinea pig. Subsequently Bast and Anson and co-workers[2-8] have made a number of contributions on the anatomical features of this organ. These authors have also strongly supported the view that the sac and the adjacent portion of the duct in which the walls have a rugose character have a resorptive function.

Some experimental studies[9-11] have suggested that a circulation of endolymph exists towards the endolymphatic sac. Other observastions[12] have indicated that active absorption takes place in the cochlear duct and that if a current is present towards the endolymphatic duct it is "without great significance for the resorption of low molecular substances in physiological concentrations within the cochlea."

In 1947 a series of experiments was reported by one of us[13] which demonstrated that the endolymphatic duct and sac were not essential to maintenance of a normal quantity of endolymph in the monkey. In three ears out of a series of seven operated the sac and medial dilated portion of the duct with its convoluted epithelium were completely destroyed. At periods of as long as three and three-quarters months no histological change in the sense organs or other labyrinthine structures was demonstrable as a result of the experimental procedures.

In order to determine if a longer postoperative period might still result in changes either in function or in histological appearance of the intralabyrinthine structures another series of experiments has been carried out.

The operative approach in the case of the monkey had been made through the mastoid and did not permit complete visualization of the actual destruction of the sac or duct and thereby resulted in many failures. Attempts made by others to repeat the experiments[8] also were reported to have failed.

In the present series the cat was used and the approach made by way of the posterior cranial fossa. The endolymphatic sac can thus be exposed and removed along with part of the duct under direct observation with the dissection microscope.

Experimental Procedure and Results:

Series A.—Histopathological changes due to obliteration of the endolymphatic sac and duct. Six cat ears were operated.

Cat 1.—Pilot experiment. Operation on left ear April 6, 1951.

A paramedian scalp incision was made and the muscles reflected away from both sides of the lambdoidal ridge. Parts of the parietal, occipital and temporal bones overlying the posterior fossa were removed. Enough cerebellum was aspirated or cut away to expose the posterior part of the medial surface of the petrous bone. The small niche in this surface indicated the location of the endolymphatic sac. Both the subdural and subosteal[1] parts of the sac were destroyed with a small burr. The adjacent part of the endolymphatic duct was also destroyed. To avoid entering the vestibule it was necessary to leave a small portion of the duct in situ. The temporalis muscle was replaced and sutured and the scalp incision closed.

Because this was a pilot experiment the cat was killed one day later. Histological examination of the inner ear revealed that the endolymphatic sac was destroyed and the endolymphatic duct was removed almost to its exit from the vestibule. Bone dust filled the exposed end of the duct at that point. All other labyrinthine structures were normal.

Cat 2.—The left ear was operated on February 12, 1951. The endolymphatic sac and adjacent endolymphatic duct were destroyed with a small burr according to the method described for the previous animal.

There was no evidence of vestibular disturbance in the postoperative period.

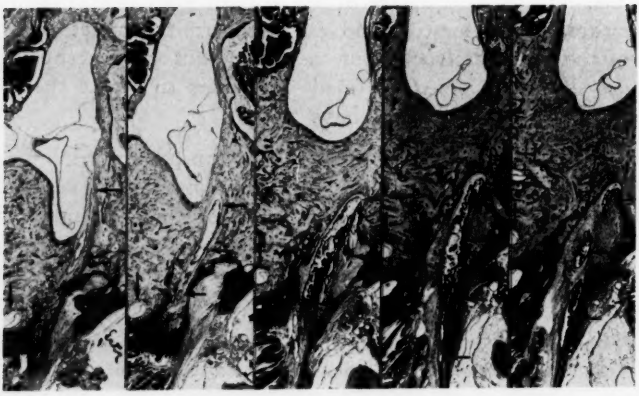

Fig. 1.—Cat 2, Right Ear. To show normal endolymphatic sac. Horizontal sections (120, 130, 150, 160, 165) through the normal endolymphatic duct and sac of the cat. The distal part of the duct adjoining the sac has a simple rugose or wrinkled epithelial lining which becomes more convoluted in the sac. The extension of the duct and sac (horizontal arrows) in relation to the position of the posterior canal (vertical arrow) serves as a reference for comparison with Figures 3, 4 and 6.

On November 19, 1951, over nine months after the operation the cat was anesthetized and perfused with Heidenhain-Susa solution. The temporal bones were sectioned 20 μ thick in the horizontal plane and every fifth section stained for histological study. Intervening sections in the region of the endolymphatic duct were stained as indicated to permit accurate studies.

Histological Examination:

Right Ear: This ear had not been operated but the sac and duct are described to permit comparison with operated ears.

Middle Ear: The middle ear was filled with purulent exudate and greatly thickened, inflamed mucosa. This type of chronic otitis media is common among the cats made available to us for experimentation.

Inner Ear: The inner ear structures were well preserved and appeared to be normal throughout.

Fig. 2.—Section through the cochlea of operated ear of Cat 2. The cochlear duct and Coti's organ appear normal throughout.

A series of five sections were selected (Fig. 1) to show the structure and extent of the endolymphatic duct and sac (horizontal arrows). In the cat the rugose character of the walls involves the whole sac as well as the adjoining dilated portion of the duct. By observing the position of the sac and duct in relation to the posterior semicircular canal (vertical arrows) and the vestibule a suitable basis is provided for comparison with the operated ears. The extent of the sac and duct which have been removed by the operation can be appreciated readily in this way.

Left Ear: Operation for obliteration of endolymphatic sac and adjacent part of duct nine months prior to death.

Middle Ear: The middle ear was filled with purulent exudate and greatly thickened inflamed mucosa.

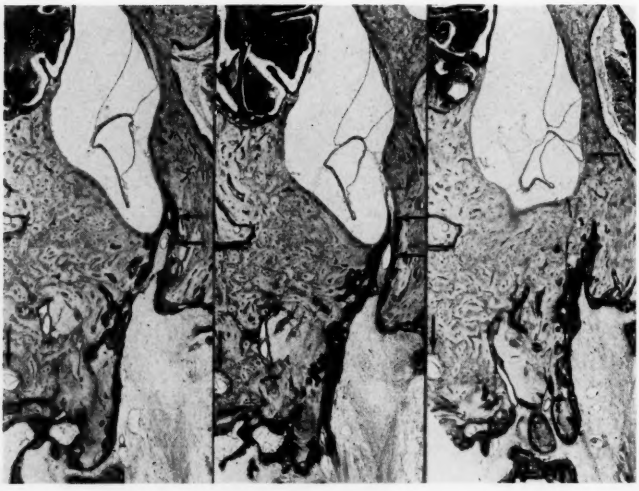

Fig. 3.—Cat 2. Operated ear. Three serial sections (35, 40, 55) to show the extent of the remaining narrow proximal part of the endolymphatic duct (horizontal arrows). Compact bone, staining darkly, has been laid down on the labyrinthine capsule bordering the operative defect, the central part of which has been filled with dense fibrous tissue. Posterior canal indicated by vertical arrows.

Inner Ear: Perilymphatic spaces normal, membranous labyrinth, utricle, saccule and cochlear duct normal in contour (Fig. 2). Receptor organs normal throughout. Sensory epithelia well preserved.

The endolymphatic sac was missing except for a tiny remnant at the outer lower extremity. This remnant was completely isolated from any connection with the labyrinth by dense fibrous tissue. Fibrous tissue and new bone filled the operative defect and extended into the vestibular aqueduct almost to its point of entrance into the vestibule (Fig. 3, horizontal arrows). The small remaining stump of endolymphatic duct was lined by a single layer of low epithelial cells. It was surounded at its outer extremity by dense fibrous tissue and compact new bone. (Compare with Fig. 1.)

Comment: Histological examination of the ear operated nine months before death revealed that the endolymphatic sac and the

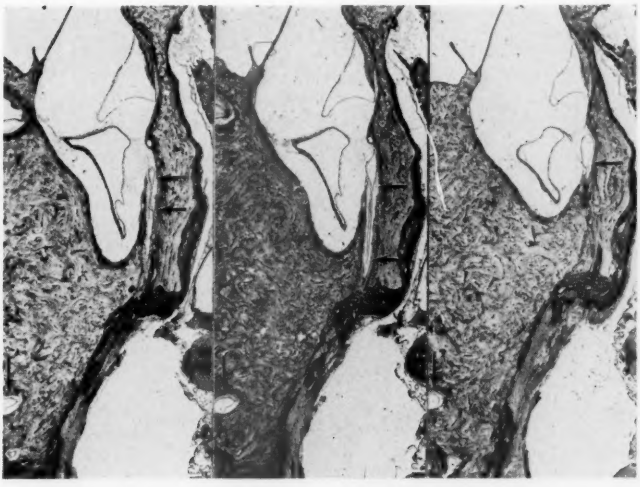

Fig. 4.—Cat 4. Right ear. Three serial sections (numbers 77, 80, 90) through the operative area showing the remaining narrow portion of the duct (horizontal arrows). The duct has healed and the aqueduct is closed off by new bone. The extent of the duct and sac which has been removed can be estimated by comparison with Figure 1, and noting the position of the posterior canal (vertical arrow) in each case.

adjacent dilated portion of the duct were destroyed and replaced by connective tissue and new bone. The other labyrinthine structures appeared normal.

Cat 3.—*Operated on March* 19, 1951. Using the technic described for Cat 1 the endolymphatic sac and duct were removed on both sides up to the point where it was feared that further drilling might open the vestibule. Postoperatively there were no signs of vestibular dysfunction. The cat was killed on November 19, 1951 (eight months later) by perfusion with Heidenhain-Susa solution. The ears were prepared for histological examination as described for Cat 2.

Middle Ears: Both middle ears were filled with purulent exudate and thickened inflamed mucosa.

Fig. 5.—Cat 4. Left ear. Mid-modiolar section through the cochlea. The receptor organs and sensory epithelia appear normal. Reissner's membrane is slightly rounded or bulged (a slight bulging or a similar degree of concavity of Reissner's membrane is sometimes seen in apparently normal specimens).

Inner Ears: On one side there was slight displacement of Reissner's membrane. This was toward the scala vestibuli similar but to a lesser extent than that shown in Figure 5. There was also some scattered amorphous exudate in the scala tympani of the basal coil. In the other ear Reissner's membrane was displaced slightly toward the cochlear duct. The appearance of Reissner's membrane in both ears is considered to be within the range of normal variation for perfused specimens.

The endolymphatic sac and the adjacent dilated part of the duct was absent on both sides. More of the duct remained in these ears than in the left ear of Cat 2 (Fig. 3). In both ears the amputated end of the endolymphatic duct was surrounded by dense new bone.

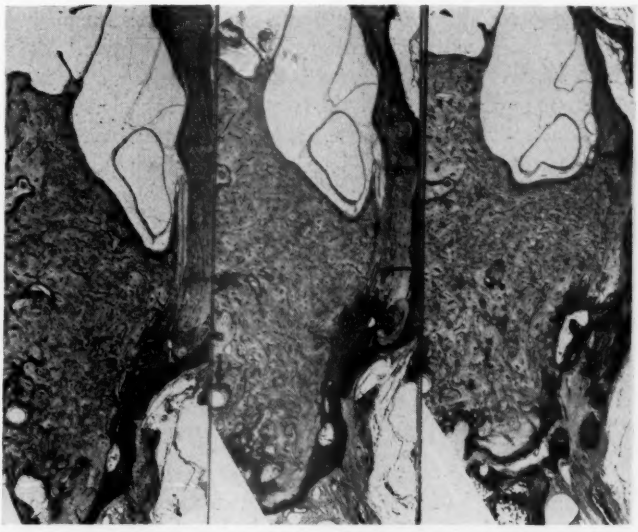

Fig. 6.—Cat 4. Left ear. Three serial sections (numbers 50, 60 and 70) through the remains of the endolymphatic duct. Only a part of the healed narrow proximal portion remains. The distal dilated portion as well as the sac are absent and the operative defect in the aqueduct has been filled with dense bone. A small remnant of the sac is present at the lower right corner but has been isolated by dense fibrous tissue.

Comment: The operations on these two ears produced almost identical injuries. The endolymphatic sac and the dilated part of the duct were destroyed. The bony channels (vestibular aqueduct) which remained were filled with new bone. The labyrinths appeared otherwise to have retained the normal histological characteristics eight months after the operation.

Cat 4.—*Operation March* 26, 1951. Bilateral destruction of endolymphatic sac and duct by the method already described. No signs of vestibular disturbance in the postoperative period. Cat killed by perfusion with Heidenhain-Susa solution November 19, 1951. Duration of experiment seven months and twenty-four days.

Right Ear:

Middle Ear: Normal.

Inner Ear: Perilymphatic spaces, membranous labyrinth, utricle, saccule, cochlear duct and receptor organs appeared normal. Reissner's membrane in cross section appears as a straight line. The endolymphatic sac was absent, the area being occupied by a layer of new compact bone on the surface of the labyrinthine capsule. The narrow portion of the duct adjacent to the vestibule was present (Fig. 4, horizontal arrows) but the dilated portion which adjoins the sac was absent. The vestibular aqueduct at its dural end was filled by dense new bone.

Left Ear:

Middle Ear: Normal.

Inner Ear: Perilymphatic spaces, membranous labyrinth, utricle, saccule, and receptor organs normal in appearance. Reissner's membrane is displaced slightly toward the scala vestibuli (Fig. 5). (Considered to be within the range of normal variation for perfused specimens.)

The endolymphatic sac is missing except for a small collapsed remnant (Fig. 6, lower right corner) which is isolated from the duct by dense fibrous tissue. The narrow portion of the duct remains (horizontal arrows). The vestibular aqueduct is filled with compact bone.

Comment: In both ears the endolymphatic sac and the distal dilated portion of the duct were destroyed and the vestibular aqueduct filled with new bone. After seven months and twenty-four days the labyrinthine structures remained histologically normal.

Series B.—Effect on hearing of obliteration of the endolymphatic sac in the cat.

This series of experiments is not completed and therefore is presented as a preliminary report. The purpose of this experiment is to determine the effect on hearing of destroying the endolymphatic sac.

Auditory testing has been completed on three animals and histological studies have been completed on one ear. A detailed protocol for one of the animals follows:

Cat 5.—(a) As a first step, the function of one ear was destroyed by macerating the labyrinth with a probe introduced through

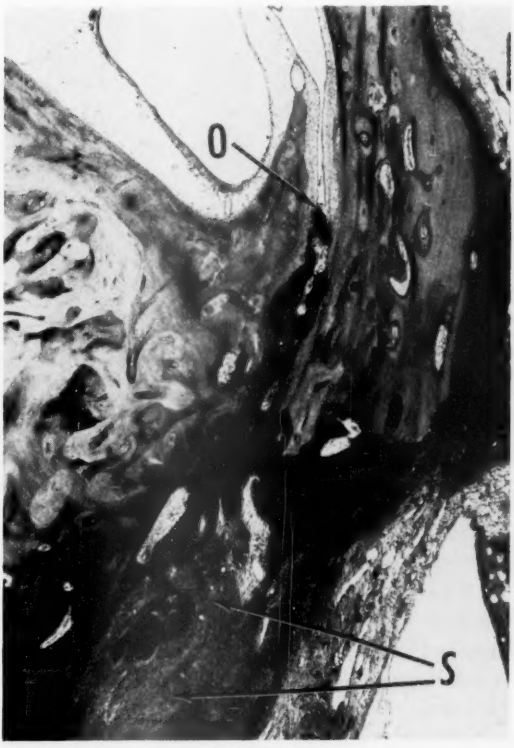

Fig. 7.—Cat 5. Photomicrograph showing new bone and connective tissue filling the site of the destroyed endolymphatic sac (s). A plug of osteoid tissue (o) has invaded the vestibular aqueduct.

the round window. After recovery from the vestibular upset which follows this procedure, the animal was trained to respond to pure tone auditory stimuli by running forward in a rotating cage to avoid shock. Using this conditioned avoidance response as an indicator of hearing, audiograms were then determined. (The method is indicated in more detail by Neff[15] and also Schuknecht, Neff and Perlman[16]). Several preoperative audiograms were determined for the animal.

Fig. 8.—Serial sections of the same ear 40μ apart to show the osteid tissue blocking the amputated end of the endolymphatic duct.

(b) On May 15, 1951 (3 months later) the left endolymphatic sac was destroyed with the dental drill as described for previous animals. A small portion of the cerebellum was removed to provide adequate exposure. The animal suffered some loss of motor co-ordination; however, this did not interfere with auditory testing. Eye movements were of a pendular type without evidence of a fast or slow component.

(c) After a period of one month auditory testing was resumed and the hearing was found to be within normal limits. On August 1, 1951 (2½ months) the animal was sacrificed under nembutal anesthesia by arterial perfusion with Heidenhain's-Susa.

The final audiogram is recorded in decibels as the difference between the preoperative and postoperative thresholds. This data appears in Table 1 and shows that no significant hearing change has occurred. On the Table the + sign before a number indicates that auditory acuity was better and the — sign that it was poorer than before destruction of the endolymphatic sac.

TABLE I.

FREQUENCY TESTED	THRESHOLD CHANGE IN DECIBELS		
	Cat 5	Cat 6	Cat 7
70	—8	+2.5	+2
125	0	+6	+1
250	—5	+2	+2
500	—6	+5	0
1,000	—4	+10	+2
2,000	—4	+7	+1
4,000	—4	—2	+4
8,000	+5	+1	—4
16,000	+10	+4	—1

(d) Histological examination shows the endolymphatic sac and the adjacent part of the duct to have been completely destroyed and replaced by a mass of young connective tissue and new bone. Osteoid tissue can be seen to extend into the vestibular aqueduct and to block the end of the endolymphatic duct. (See Figs. 7 and 8.) The remainder of the inner ear structures appear normal. The hair cells are normal and Reissner's membrane is not displaced. The middle ear is free of pathological change.

Cats 6 and 7.—The same experimental procedures were followed as in Cat 1. Table 1 shows that for both animals auditory function was within normal limits four months after obliteration of the endolymphatic sac. The inner ears are now in the process of histological preparation.

Comment: Destruction of the endolymphatic sac and the adjacent dilated portion of the duct failed to produce a change in auditory acuity in three animals. The time lapse was two and one-half months in one animal and four months in the other two animals. Histological sections of the inner ear from one of these demonstrates complete obliteration of the endolymphatic sac and part of the duct with blockage of the amputated end of the endolymphatic duct. There are no other changes in this ear.

PART II.—COCHLEAR AQUEDUCT AND PERIOTIC DUCT

We will use the term "cochlear aqueduct" to refer to the bony channel lying within the otic capsule extending from the scala tympani to the cranial cavity. The term "periotic duct" will designate the tube-shaped structure of arachnoid-like tissue and fluid which fills the cochlear aqueduct.[17]

It has been generally accepted that fluid can pass readily through the meshwork of the periotic duct. Thus it has been suggested by some that the main source of perilymphatic fluid is from the passage of subarachnoid fluid through the periotic duct into the scala tympani. Acceptance of this idea is complicated by the report that blockage of the cochlear aqueduct can occur without histological changes in the inner ear.[18] Altmann and Waltner[19] studied the problem of fluid flow by injecting iron salt solutions into the subarachnoid space after the method of Weed. They were unable to demonstrate a fluid current through the periotic duct. The question is still not settled as to whether particulate matter can pass through the periotic duct.[20, 21]

The experimental study of Uyama[22] is of particular interest in relation to our own investigation. He used two methods for blocking the cochlear aqueduct in the rabbit. One method used was to inject a viable suspension of sarcoma tissue into the subarachnoid space near the cranial opening of the cochlear aqueduct. Histological studies revealed that the sarcoma cells had proliferated and filled the lumen of the cochlear aqueduct in some animals. The second method consisted of introducing a fine piece of catgut into cranial end of the cochlear aqueduct. There was histological proof that the catgut had blocked the channel completely in some animals.

In most of the ears in which the cochlear aqueduct was blocked he found displacement of Reissner's membrane toward the scala vestibuli. He also reported finding a glassy precipitate in the scala tympani in these ears. These findings are shown in the microphotographs which accompany the report. He reasoned that the displacement of Reissner's membrane toward the scala vestibuli was due to a decrease in volume of perilymphatic fluid and a compensatory increase in endolymphatic fluid to take up the space. He concluded on this basis that perilymphatic fluid was derived from cerebrospinal fluid which passed through the cochlear aqueduct.

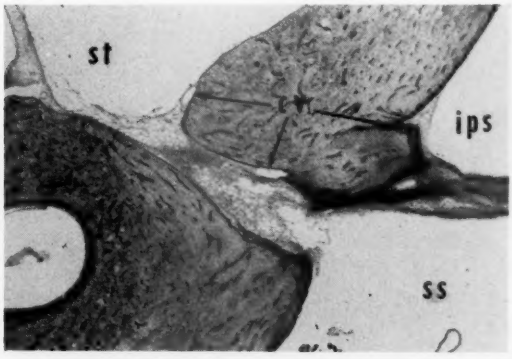

Fig. 9.—View of normal cochlear aqueduct and its contained periotic duct in the cat. S.T.—scala tympani, S.S.—subarachnoid space, C.V.—cochlear vein, I.P.S.—inferior petrosal sinus.

The cochlear aqueduct and its contained periotic duct have a wider lumen in most quadrupeds than in humans (See Fig. 9). In the cat it is about two mm in length. The size of its lumen decreases progressively from the cranial end toward the scala tympani where it widens again. At its narrowest point it is about .3mm in diameter.

Most of the venous return from the cochlea passes through the cochlear vein. This vein courses along the wall of the scala tympani and enters the cochlear aqueduct. Usually before doing so it is joined by a small vein which originates in the mucosa of the auditory bulla and reaches the scala tympani by passing through the round window membrane. For a distance of about 1.2 mm the cochlear vein lies within the cochlear aqueduct adjacent to the periotic duct. At this point it enters a separate bony channel and continues within it to the inferior petrosal sinus.

The purpose of this experiment was to determine the effect of blocking the periotic duct on the function of the inner ear. It would be of considerable interest, for example, if one could reproduce Uyama's experimental findings, namely, endolymphatic hydrops following blockage of the periotic duct.

Our experiments on the periotic duct are incomplete and are presented here as a preliminary report.

Auditory testing and histological studies have been completed on two animals.

Experimental Method and Results:

Cat 8.—(a) On Aug. 20, 1950, a left destructive labyrinthotomy was performed. Auditory conditioning was accomplished during the following three months.

(b) On Nov. 21, 1950, an operation was performed on the right ear to interrupt the cochlear aqueduct. The auditory bulla was exposed and a cap of bone removed. The method for removing and replacing this bony cap is described in detail elsewhere.[17] The mucosal lining was reflected from the superior wall of the auditory bulla in the region immediately posterior and medial to the round window. By using a very small dental burr to remove bone adjacent to the cochlear wall, it was possible to enter the cochlear aqueduct from its ventral side without opening the labyrinth in any other area. On approaching the cochlear aqueduct the intervening bone became very thin so that the cochlear veing could be seen within it as a red line (under magnification). The cochlear aqueduct was opened widely. The cochlear vein was injured and bled freely. Bone dust was packed into the opened ends of the aqueduct. The previously prepared flap of middle ear mucosa was laid down into the surgical defect on top of the bone dust. The cap of bone which had been removed from the bulla was wired back into place.

(c) After three months auditory testing was resumed. The thresholds were found not to be significantly changed. Table II shows the differences in threshold between the pre-operative and the final postoperative tests. The + sign indicates better sensitivity and — sign poorer sensitivity in the final test.

(d) On July 20, 1951 (eight months) the animals auditory acuity was still within normal limits. Because of the time and work involved in conditioning these animals for auditory testing, it was decided to attempt a partial eighth cranial nerve section by an approach from the posterior fossa. The cochlea was accidentally entered during the operation. Auditory tests during the following week revealed the animal to be totally deaf.

(e) On August 22, 1951, the animal was sacrified under nembutal anesthesia by arterial perfusion with Heidenhai-Susa.

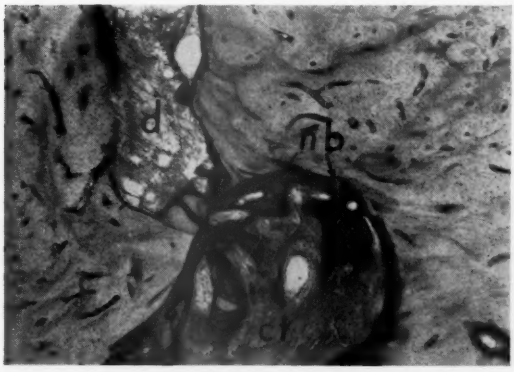

Fig. 10.—Cat 8. High power view of the cochlear aqueduct and periotic duct (D) showing connective tissue (C.T.) and compact new bone (N.B.) blocking its lumen.

The cochlea was serially sectioned in the usual manner and every fifth section was stained and mounted. Additional sections were prepared in the area of the cochlear aqueduct to allow a more detailed study. Camera lucida tracings were made from the serial sections so as to better define the relationship of new bone and connective tissue to the periotic duct and to establish the fate of the cochlear vein.

The operative lesion was found to intercept the periotic duct at a point one mm from the vestibular end. The lesion consisted of vascular connective tissue in its center (See Fig. 10). The margins were lined with compact new bone. No connections could be found to exist between fluid spaces of the periotic duct and the fluid and vascular spaces of the tissue which had filled in the operative defect.

The cochlear vein connected with venous channels in the new connective tissue, the latter having connections with the veins in the mucosa of the auditory bulla.

The cochlea of this animal was severely damaged. The scalae were partially filled with young connective tissue and the sensory and neural elements were severely degenerated. (See section d. for explanation.)

TABLE II.

FREQUENCY TESTED	THRESHOLD CHANGE IN DECIBELS	
	Cat 8	Cat 9
70	+1	—1
125	+7	—4
250	—2	—15
500	—3	—10
1,000	—5	—9
2,000	—5	0
4,000	+2	0
8,000	+15	—7
16,000	0	—4

Cat 9.—(a) The same experimental procedure was followed as for Cat 1. The animal was sacrificed four months after the operation for interruption of the periotic duct. His auditory acuity remained within normal limits during that period (See Table II).

(b) Examination of serial sections showed complete interruption of the fluid spaces in the periotic duct by new bone and connective tissue. The cochlear vein had recanalized through this tissue and connected with the inferior petrosal sinus.

The cochlea appeared normal. Reissner's membrane was in its normal position.

Comment: An operation to interrupt the periotic duct was performed in two animals. There was no change in auditory acuity following this procedure for a period of eight months in one and 4 months in the other. There was no evidence of endolymphatic hydrops in Cat 9 whose cochlea was available for study. Thus the findings in the one cochlea available for study do not support the observations of Uyama[22] It may be that the endolymphatic hydrops occurring in his animals was due to a mild labyrinthitis caused by the foreign materials used to block the cochlear aqueduct.

COMMENTS AND CONCLUSIONS

The anatomical features of the endolymphatic sac suggest that this organ may have an important resorptive function. If it has such an action which is vital to the functional integrity of the inner ear, then destroying it should create histological changes and deafness.

One of us (J.R.L.) previously reported that obliteration of the endolymphatic sac in the monkey produced no histological changes of the cochlea over a period of three and one-half months. The experiment has been repeated on seven cats, three of which were conditioned for auditory testing. The time lapse in different animals varied from three to nine months. Serial sections of the cochleas failed to show significant histological changes. The auditory thresholds of three cats tested were not significantly changed by the operation.

The periotic duct, which lies within the cochlear aqueduct, provides an intimate relationship between the perilymph of the scala tympani and the cerebrospinal fluid of the subarachnoid space. Some studies have shown that there is a slow passage of cerebrospinal fluid through the duct into the scala tympani. On two cats we performed surgical procedures by which the periotic duct was successfully blocked. There were no histological changes in the inner ears or significant changes in auditory acuity with time lapses of four and eight months.

The experimental results show that the inner ear suffers no histological or functional injury by losing the services of the endolymphatic sac or periotic duct. These should not be interpreted to indicate that these structures perform no function. Some service may be performed which can be assumed by other structures in their absence. Nevertheless the evidence indicates that, in the cat and monkey, obliteration of either the endolymphatic duct or the periotic duct is not in itself a cause of labyrinthine disease.

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(Discussion appears on page 738)

LXIV

NEW THEORY FOR THE CORRELATION OF THE PATHOLOGY AND THE SYMPTOMATOLOGY OF MENIERE'S DISEASE

A RESEARCH STUDY OF THE VESTIBULAR ENDOLYMPHATIC LABYRINTH

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Though much has been studied, theorized and written about the etiology and pathology of Meniere's Disease, it nevertheless has to be admitted that a much more logical answer in explanation of this baffling but most interesting symptom complex than has heretofore been offered will have to be furnished before this problem can be considered as finally solved.

Ever since Hallpike and Cairns[1] first reported that distention of the cochlear duct and absence of the normal soft perisaccular connective tissue surrounding the saccus endolymphaticus were the most significant pathological post-mortem findings within the labyrinths of two patients whose eighth nerve was sectioned for the relief of Meniere's symptom complex, it has been more or less generally accepted that Meniere's symptom complex is caused by either hyper-

From Lempert Institute of Otology, Lempert Research Foundation, Inc.

secretion or poor resorption of endolymph. These pathologic findings have since been corroborated by other otologists. Meniere's Symptom Complex is therefore now predominantly referred to in the literature as Endolymphatic Hydrops. Despite the fact that Hallpike's interpretation of the pathological findings in the labyrinth is essentially correct, I was never satisfied with his or anyone else's raison d'etre for these findings or their attempts at correlating these findings to the symptom complex.

Outside of cochlear duct distention being observed repeatedly post-mortem in patients with Meniere's Disease, no concrete histological evidence has yet been demonstrated that the hydrops within the ductus cochlearis actually represents either a hypersection of endolymph or an impeded resorption of normally secreted endolymph or both a hypersecretion and impeded resorption of endolymph.

On the contrary if Guild's assumption that endolymph is secreted by the stria vascularis is correct, the not infrequent finding of atrophy of the stria vascularis in the presence of cochlear duct distention would tend to disprove the theory that the distention of the cochlear duct is the result of hypersection of endolymph. The fact that both the presence of normal and atrophic perisaccular connective tissue have been observed in the presence of cochlear duct distention and also the fact that atrophic perisaccular tissue has been observed in an otherwise normal ear would tend to discredit the theory that the cochlear duct distention is strictly the result of impeded resorption of the normally secreted endolymph.

Most disappointing and disconcerting to me was the fact that in spite of the nondisputable presence of histological evidence of hydrops within the cochlear duct as evidenced by the frequently found distention and occasional rupture of Reissner's Membrane, the clinical picture of Meniere's symptom complex could nevertheless not be logically correlated to the cochlear duct hydrops on the basis of increased pressure of endolymph hypersecretion being the cause thereof.

No one as yet has satisfactorily explained why in the presence of histologic evidence of increased endolymph pressure sufficient to cause gross dilatation of the cochlear duct and displacement of Reissner's Membrane plus some dilatation of the saccule, neither the membranous semicircular canals nor the utricle show any evidence of distention. Hallpike says that certain portions of the normal utricular

Fig. 1.—Photomicrograph. Normal membranous semicircular canal with normal epithelial lining autopsy of a child of two years. Courtesy of Dr. Eggston. No. 4 Section 528.

Fig. 2.—Photomicrograph. Normal and vesicular areas within the membranous external canal wall removed from a patient during partial labyrinthectomy for Meniere's Disease. White male, 60 years. No. 2490 Slide 8a-2.

walls are perhaps two or three times as thick as those of the rest of the utricular walls and therefore the utricle does not become dilated. If this were true then the thinner portions of the utricular walls would give way to the endolymph pressure and show signs of localized distention within the otherwise nondistended utricle.

No one so far as I know has as yet logically explained how increased endolymphatic pressure within the cochlear duct and saccule only can be held responsible for the severe vertigo which accompanies the paroxysmal attacks of Meniere's Disease. If increased pressure involved the entire vestibular labyrinth it would result in hypoactivity of the crista. The argument given that such hypoactivity when reaching a stage of temporary paralysis of the crista could give rise to hyperactivity of the crista in the other ear wherein the endolymph pressure is normal, cannot be supported as long as histologic evidence of increased endolymph pressure within the utricle and semicircular canals in the diseased ear cannot be seen.

The paroxysmal nature of the attacks in Meniere's Disease is still to be explained. No one has as yet shown that if hypersecretion of endolymph is the cause of the disease why the vertigo reaches the height of its intensity only during these paroxysms. Neither has anyone explained how poor absorption of endolymph due to atrophy of perisaccular tissue can be held responsible for Meniere's symptom complex occurring in paroxysms.

Rollins[2] explanation that the loss of hearing in Meniere's Disease which progresses with each paroxysmal attack is due to the distended saccule pressing against the labyrinthine surface of the stapes cannot be supported in the face of existing experimental evidence. Bekesy[3] has experimentally proven that increased intralabyrinthine pressure does not interfere with the mobility of the stapes. Wever, Lawrence, Meltzer and I[4] have proven that increased pressure within the labyrinth does not interfere with the cochlear potentials. Furthermore, the permanent hearing loss in Meniere's Disease is unquestionably a perceptive loss and not a conductive loss of hearing.

The fact that in 3 cases of Meniere's Disease I failed to relieve the attacks of vertigo by destruction of the cochlear duct is further proof that endolymphatic hydrops pressure is not the cause of the vertigo. Subsequent destruction of the membranous external semicircular canal stopped the attacks of vertigo.

Fig. 3.—Photomicrograph. Vesicles seen in the longitudinal cut of the membranous canal. A fragment of a cystic macula lies in the adjacent collapsed portion of the membranous labyrinth. White male, 57 years. No. 1905 Slide 10 Section 2.

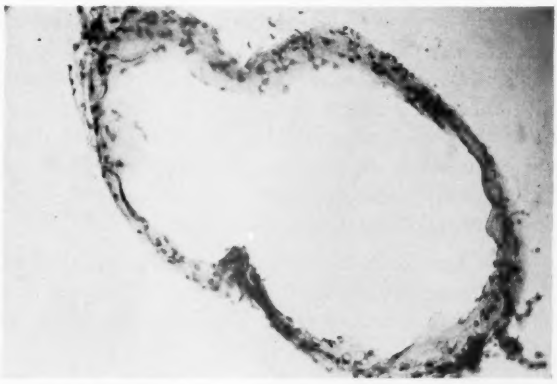

Fig. 4.—Photomicrograph. Cross section of a membranous semicircular canal showing (a) epithelial vesicles (b) globules attached to parent nuclei. These coalesce to form the protruding vesicles. White male, 43 years. No. 1955 Slide 5.

Furthermore if endolymph hydrops pressure were the cause of Meniere's symptom complex, how come that section of the vestibular fibers of the eighth nerve stops the vertigo without affecting the residual hearing, despite the fact that it does nothing to relieve pressure within the cochlear duct from endolymph hypersecretion or to improve resorption?

In view of the above mentioned inconsistencies between Meniere's symptom complex and the histopathology to which these symptoms are attributed, it seems quite obvious to me that neither endolymph hypersecretion nor poor resorption of normal endolymph secretion can be logically held responsible for Meniere's symptom complex.

The above mentioned facts suggested to me the possibility that Meniere's Disease may primarily be a disease of the vestibular labyrinth and that the cochlear pathology and symptoms are the sequelae of the vestibular disease.

I decided to make an extensive histological study of the vestibular membranous labyrinth removed in vivo during the performance of partial labyrinthectomy for the arrest of vertigo in Meniere's Disease in order to determine if possible whether the vestibular labyrinth is playing perhaps a greater part in producing Meniere's symptom complex than it is being credited with.

I performed a partial labyrinthectomy in six consecutive patients suffering from repeated severe paroxysmal attacks of Meniere's symptom complex for the relief of the vertigo. I carefully removed the membranous endolymphatic external semicircular canal including the ampulla with the least amount of trauma in every one of these six cases. Part of the utricle was pulled along with four of these canals. I gave these immediately to my associate, Dorothy Wolff, who preserved and sectioned these specimens in celloidin for high power microscopic examination in the usual manner.

So far as we know, the histopathology of the membranous labyrinth or portions thereof removed from living patients during the performance of labyrinthectomy for Meniere's Disease has not been reported.

RESUME OF THE HISTOLOGIC OBSERVATIONS MADE WITHIN THE THE BIOPSIED MEMBRANOUS EXTERNAL SEMICIRCULAR CANALS

Maculae from four patients and a crista from two were observed. The end-organs of the human canals seen, all showed marked de-

Fig. 5.— Photomicrograph. A portion of a membranous semicircular canal showing (a) normal and vesiculated areas within its inner lining (b) mass rupture of vesicles into the lumen, due to low surface tension of the vesicular wall. White male, 28 years. No. 1935 Slide 7.

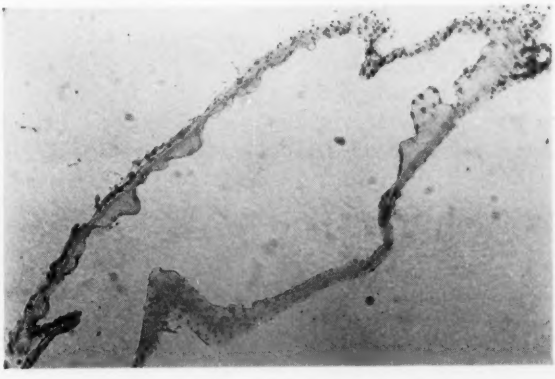

Fig. 6.—Photomicrograph. Vesicles appear along the straight portions of a canal and not at points of mechanical distortion. Note the clear globules attached to nuclei. White male, 49 years. No. 2547 Slide 14.

generative changes of a chronic type. One crista observed showed atrophic epithelial surface with no evidence of hair cells and atrophic nerve fibers. The other crista showed round cell infiltration. In one macula in which the hair cells looked like ciliated epithelium two phleboliths were apparent on the surface. Marked edema along nerve fibers was apparent in two maculae and atrophy of nerve fibers present. Two maculae showed cystic degeneration, the large cysts being filled with floccular substance. A brown granular pigment was abundant in these cystic areas. Whether this is melanin or hematoidin or hemosiderin could not be determined but it did not appear in vascular channels, so presumably it is melanin.

A histologic study of the membranous walls of the six endolymphatic external semicircular canals was made. Instead of the uniformly smooth histologic structure of the inner lining of the membranous semicircular canals which is observed post-mortem within the normal temporal bone (Fig. 1), the inner lining of the six membranous external semicircular canals removed in vivo from six consecutive patients suffering from Meniere's Disease during the performance of partial labyrinthectomy was invariably found to be studded with vesiculated epithelial excrescenses varying in size and number suggestive of the formation of epithelial fluid collections (Figs. 2-7 inclusive). In order to exclude the possibility of these excrescenses in the inner lining having been produced by the surgical trauma, my associate, Dr. Rambo removed the membranous external semicircular canals from two Rhesus monkeys in vivo and gave them immediately to Dorothy Wolff who preserved and sectioned these specimens in celloidin for high power microscopic examination in the usual manner. The inner lining of the membranous walls showed no vesiculation in any of the biopsied monkey labyrinths.

In the October 1938 issue of the Journal of Laryngology and Otology under the title of "Observations on the Pathology of Meniere's Syndrome," Hallpike[1] states as follows:

"It is the purpose of this paper to describe—it is believed for the first time—the pathological changes in the temporal bones of two cases of Meniere's Syndrome. In both, death occurred shortly after operation for section of the VIIIth Nerve."

Hallpike described the post-mortem histological findings within the semicircular canals of Case One as follows:

Fig. 7.—Photomicrograph. Four areas of vesiculation occurring along the lumen of a canal in no relation to the sharp bend. Stained in Masson's trichrome. White female 65 years. No. 2620 Slide 3a section 3.

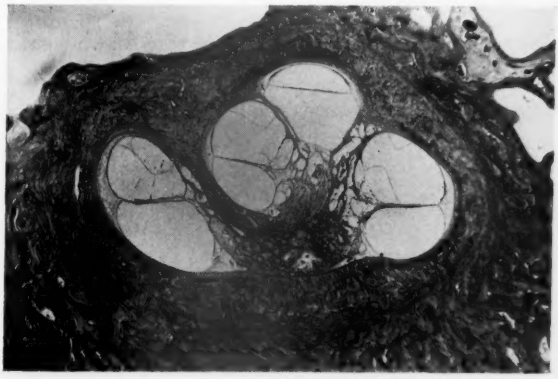

Fig. 8.—Photomicrograph. So-called hydrops of the human cochlea from a case diagnosed as Meniere's Disease before death. Note atrophy of stria vascularis, Corti's organ and the spiral ganglion cells. Washington University collection. No. 5178 Left Section 1062.

"In the internal limb of the anterior vertical canal and in the lateral canal certain changes are present in the lining epithelium which are reproduced in Fig. 2. The epithelium has been raised in localized excrescenses apparently by the formation of sub-epithelial fluid collections."

In his description of the post-mortem histological findings within the semicircular canals of Case Two he stated as follows:

"The lateral canal shows a very marked formation of subepithelial vesicles. Fig. 22."

Since Hallpike also observed what he calls subepithelial vesicles in 7 out of 10 cases between the ages of 40 and 60 years, without a history of Meniere's Disease, he stated as follows:

"Its pathological significance, meaning subepithelial vesiculation, must therefore be regarded as doubtful."

In 1943 Altmann and Fowler, Jr.[5] corroborated the findings of Hallpike in three of their own cases of Meniere's symptom complex. They called attention to having observed epithelial vesiculation in two of their three cases. In describing the histologic findings in the right ear of Case Two they stated as follows:

"Some epithelial vesiculation is present in the semicircular canals and in the utricle and saccule."

They also stated that this condition is of doubtful pathological significance.

Dorothy Wolff had processed in 1937-38 the temporal bones from a case diagnosed as Meniere's Disease while at Washington University Medical School. This case is illustrated in Figures 8 and 9. We corroborate Hallpike's findings of vesiculation of the membranous semicircular canals and cochlear duct distention in Meniere's Disease. It occurred to me, however, that if, as Hallpike states, subepithelial vesiculation within the membranous semicircular canals has been observed also in routine postmortem human material between the ages of 40 and 60 in the absence of a history of Meniere's Syndrome, then this in itself would not be sufficient to dismiss this histological finding as of doubtful pathological significance in either instance.

I reasoned that if epithelial vesiculation within the membranous semicircular canals is of no pathological significance and represents

Fig. 9.—Photomicrograph. Chronic vesiculation in a membranous semicircular canal from a patient with a diagnosis of Meniere's Syndrome. Washington University collection. No. 5178 Right. Section 534. Post mortem.

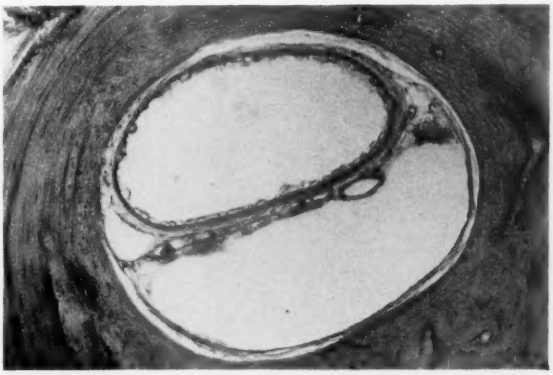

Fig. 10.—Photomicrograph. Medial limb of left superior semicircular canal of the monkey. Note series of vesicles, one exuding above each nucleus of the epithelium. No. 13 Left, Section 140.

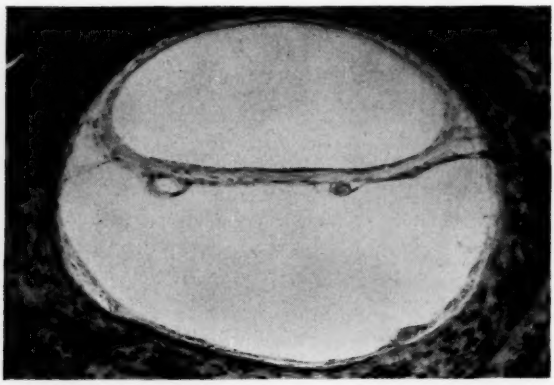

Fig. 11.—Photomicrograph. Detail showing normal epithelium in medial limb of superior membranous semicircular canal in the right ear of the monkey with the normal cochlea. No. 13 right section 150.

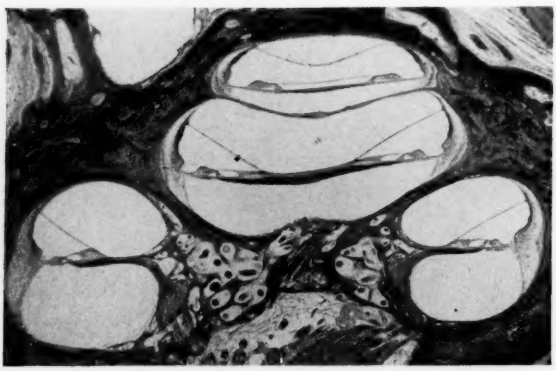

Fig. 12.—Photomicrograph. Normal right cochlea from the ear of a fenestrated monkey. Note the normal position of Reissner's membrane in each coil of the cochlea. No. 13 Right Section 210.

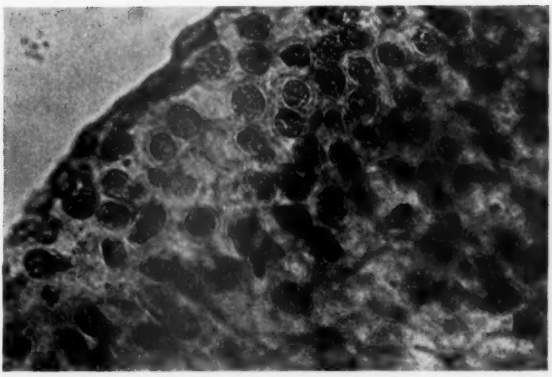

Fig. 13.—Photomicrograph. Surface view of normal epithelium overlying the interior of the collagenous membranous semicircular canal wall. Each nucleus is surrounded by a clear halo which indicates the production of the column of thickened basement membrane produced by it to form the collagenous wall illustrated in Fig. 15. White, female, 65 years. No. 2620 Slide 26 Section 1.

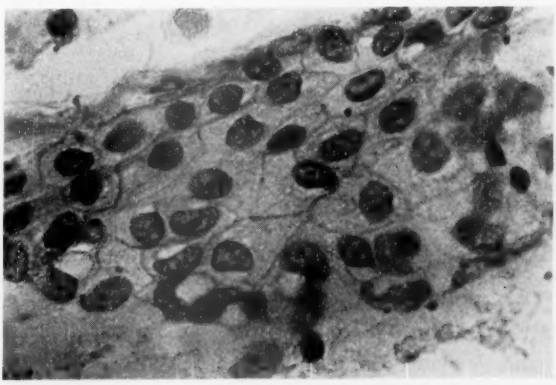

Fig. 14.—Photomicrograph. Relatively normal epithelial cells of the inner wall of a membranous semicircular canal. Eight or nine cells show pathological globules. White male, 49 years. No. 2547 Slide 11.

the normal histologic structure of the vestibular endolymphatic labyrinth, then why it is not constantly observable in all post-mortem human material of all ages? Since it is mainly observed between the ages of 40 to 60 years, is it not more plausible to interpret it rather as a chronic progressive degenerative change within the membranous labyrinth which has taken place in vivo? Perhaps this epithelial vesiculation which Hallpike observed between the ages of 40 to 60 years may have something to do with the common vertigo so frequent in people of that age. If epithelial vesicultion represents a post-mortem change, then why is it as a rule not observed post-mortem in infants and only rarely in the adult under the age of 40 years? The fact that epithelial vesiculation was found in biopsied material as well as in autopsy material is sufficient proof that the epithelial vesiculation observed is not the result of post-mortem degeneration per se. By the same token the fact that it is repeatedly observed in autopsied material of patients known to have suffered from Meniere's Disease without having had any otologic surgery is further proof that the epithelial vesiculation observed in our six biopsied specimens from living Meniere's Disease sufferers is not the result of surgical trauma. Furthermore the fact that it is often observed post-mortem to exist unilaterally instead of bilaterally is proof sufficient that vesiculation is not a normal histologic feature. It is also noteworthy that epithelial vesiculation within the vestibular labyrinth of the Rhesus monkey has been observed only in one out of more than a hundred experimentally fenestrated Rhesus monkey ears. Although both of that monkey's ears were fenestrated, epithelial vesiculation was found only in one of the two ears (Figs. 10, 11, 12). This histologic finding in the left ear, plus the fact that the right ear in that same monkey showed no sign of epithelial vesiculation is in itself sufficient proof that epithelial vesiculation when present cannot be considered as a normal finding and devoid of pathological significance.

In view of the fact that despite the findings of cochlear duct dilatation which is suggestive of endolymph hydrops there exists no histological evidence to prove that either hypersecretion or poor resorption of endolymph actually takes place in Meniere's Disease and since Meniere's symptom complex cannot be correlated logically to endolymphatic hydrops without stretching dangerously one's imagination, it occurred to me that perhaps the epithelial vesiculation which has been so readily dismissed as of doubtful pathological sig-

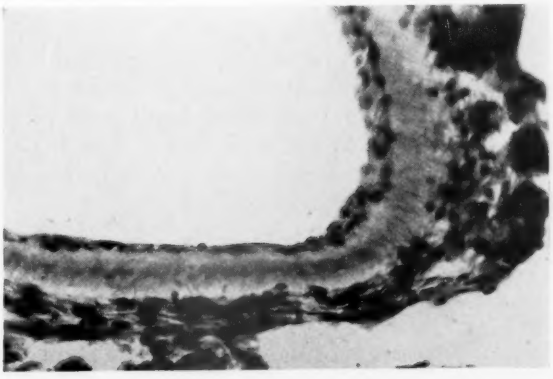

Fig. 15.—Photomicrograph. A slide view of a membranous semicircular canal showing epithelium subtending collagen columns of a canal wall. White female, 65 years. No. 2620 Slide 26a Section 1.

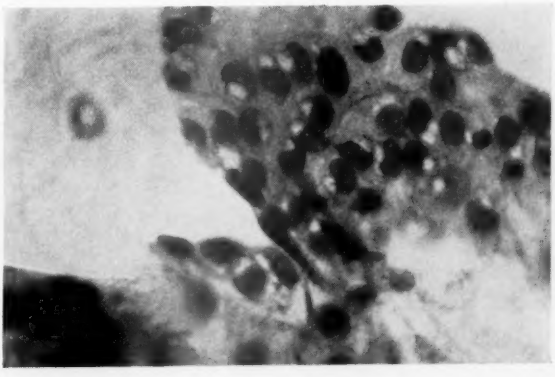

Fig. 16.—Photomicrograph. Detail of nuclei exuding single globules. One nucleus may exude two or more globules. These may coalesce to form a large vesicle. White female, 65 years. No. 2620 Slide 22 Section 2.

nificance is actually playing a greater role in Meniere's Disease than has been suspected.

In order to determine if possible whether epithelial vesiculation which we observed in our six biopsied membranous external semicircular canals is or is not of pathological significance in Meniere's Disease, I decided to carry our histologic investigation of the cellular structure in the inner lining of these membranous labyrinths a little further than it was heretofore subjected to.

SUMMARY OF OBSERVATIONS MADE IN A STUDY OF THE CELLULAR STRUCTURE IN THE BIOPSIED PORTIONS OF THE VESIBULAR LABYRINTH

The histologic structure of the inner lining of the membranous walls in every one of the six endolymphatic external semicircular canals removed from six patients with Meniere's Disease during the performance of partial labyrinthectomy reveals that the normal portion of the membranous canal bears an inner lining of delicate squamous-type epithelial cells of hexagonal shape. These squamous cells cover those areas of the inner canal wall not occupied by specialized sensory epithelium (Figs. 13 and 14).

In the normal regions it is apparent that the nuclei of these cells produce the acellular collagenous substance which comprises the thickness of the membranous semicircular canal wall (Fig. 15). The act of production of this substance appears from the surface of the epithelium as a clear halo around the nucleus (Fig. 13). When seen from the side view this semifluid is observed to extend downwards from each cell, in columns. The columns soon lose the individuality of their discrete cellular derivatives, becoming a homogeneous mass or wall, subtended by the epithelial layer. The homogeneous substance stains faintly with eosin in H & E preparations and a brilliant blue in aniline blue collagen stains such as Mallory's and Masson's. Presumably therefore it is collagen. Immediately beneath the acellular layer of the wall runs a relatively wide, thin-walled vascular channel, whose function is undoubtedly to remove degenerative products continuously from this wall.

As interpreted from the microscopic sections after studying the histologic cellular structure in the normal areas of our biopsied specimens the following sequence of histopathologic events were observed in the vesiculated regions of the inner lining of the mem-

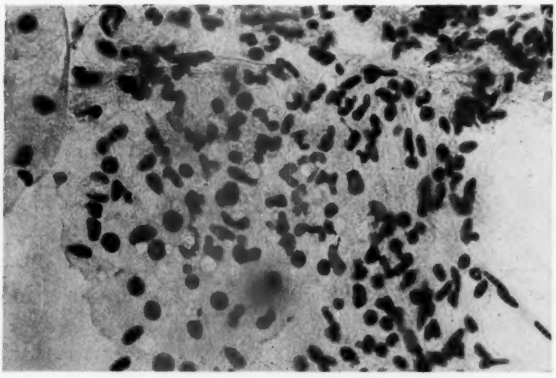

Fig. 17.—Photomicrograph. Amoeboid and cuneiform nuclei develop as globules are exuded. The nucleus is eventually exhausted and becomes necrotic. White female, 65 years. No. 2620 Slide 8.

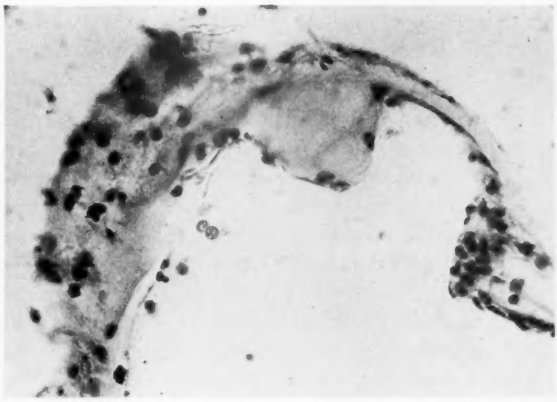

Fig. 18.—Photomicrograph. Detail of enlarged vesicle formed by coalescence of single globules. This sooner or later ruptures. White male, 43 years. No. 1955 Slide 5-4.

branous external semicircular canals. These are suggestive that the epithelial vesiculation is undoubtedly of pathological significance in Meniere's Disease.

First Pathological Stage: Each nucleus of the epithelial lining of the membranous canal begins to exude a single clear globule. This distorts the spherical shape of the nucleus so that each nucleus becomes elongated pointing toward the globule. The globule increases in size, becoming much larger than the nucleus. The nucleus apparently attempts to maintain the same relationship toward the globule that it does towards its normal production, by "capping" it. In the pathological set-up however, the substance formed is exuded toward the lumen of the canal, instead of away from it. The result is, we get a polka dot appearance over the tissue with clear circles each capped by a nucleus (Fig. 16).

Second Pathological Stage: Surface tension may break so that the clear globules burst individually and pour their contents into the lumen of the canal (Fig. 5).

Third Pathological Stage: A single nucleus may produce a second globule or perhaps three or four (Fig. 16). The second globule may be exuded beside the first and may coalesce with it or it may be exuded from the opposite side of the nucleus. Many globules coming from various points on the surface of the nucleus soon distort the contour of the nucleus so that it becomes cuniform or amoeboid in shape (Fig. 17). As with the above stage, the globules or vesicles formed may burst and pour their contents into the lumen of the canal.

Fourth Pathological Stage: The erupting globules coalesce and bulge into the lumen of the canal building up a relatively strong surface tension with chronicity (Fig. 18). As the vesicles crowd upon each other like cumulus clouds (Fig. 19a), they push their parent nuclei out of the way, either toward the lumen or more often back toward the peripheral side of the membranous wall (Fig. 19b). Here we see them as dumb-bell shaped bodies, or they may resume their round form and appear almost like a round cell infiltration process. Even this relatively strong surface tension may break (Fig. 19c) and again a substance which appears as a granular precipitate is poured into the lumen of the canal (Fig. 19d).

The pathological vesiculated substance as it accumulates loses its perfectly clear appearance and stains with eosin in H & E prep-

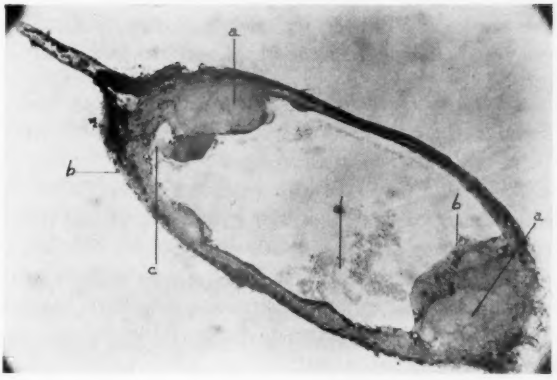

Fig. 19.—Photomicrograph. Chronic vesiculation of a membranous semicircular canal showing (a) cumulus cloud formation of vesicles, (b) nuclei being pushed centrally toward the lumen and peripherally, (c) a break in the surface tension with rupture and (d) granular precipitate within the lumen. White male, 60 years. No. 2490 Slide 2 Section 2.

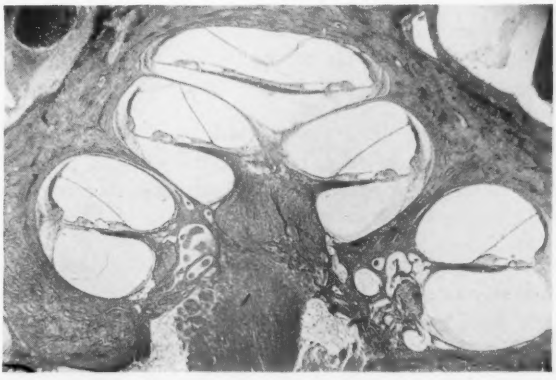

Fig. 20.—Photomicrograph. The left cochlea of the monkey whose vesiculated left membranous semicircular canal was illustrated in Figure 10. This left ear was also fenestrated. Note the area showing evidence of cochlear duct distension in the middle coil. No. 13 Left, Section 270.

arations. It stains more brilliantly blue in Masson's trichrome, even as does the normal acellular portion of the canal wall. We are thus led to believe that this substance is collagenous in nature.

The above described histopathologic events observed in the histologic cellular structure of the vesiculated areas are suggestive that the epithelial vesiculation is undoubtedly of pathological significance in Meniere's Disease. It is also our belief that epithelial vesiculation of the inner lining of the vestibular membranous labyrinth is always pathological whether or not it is accompanied by Meniere's symptom complex. We recognize the fact that the degree of symptomatology may depend upon the degree of vesiculation that has taken place. Minor degrees of vesiculation may account for the transient vertigo so common after the age of 40.

In further substantiation of our belief that epithelial vesiculation of the inner lining of the vestibular membranous labyrinth is of pathological significance in Meniere's Disease is the interesting observation that in the one and only monkey ear in which epithelial vesiculation was found, histologic evidence of cochlear duct distention was also present (Fig. 20).

NEW THEORY FOR THE CORRELATION OF THE PATHOLOGY AND THE SYMPTOMATOLOGY OF MENIERE'S DISEASE

By accepting epithelial vesiculation as part of the pathological entity in Meniere's Disease, a much more plausible correlation of Meniere's Symptom Complex to the pathology is made possible than ever before.

Based upon our observations made as a result of our research study we believe that Meniere's Disease is a chronic progressive Herpetic Neuritis of the vestibular labyrinth of either toxic or trophic origin.

We propose the theory that an attack of Meniere's Symptom Complex is due to the rupture of one or more vesicles with the release of a toxic fluid, whose chemical constitution differs from that of the normal endolymph, into the lumen of the endolymphatic membranous labyrinth. The size and number of vesicles rupturing at one time determine the severity of the attacks. The paroxysmal nature of this disease is due to the continued formation and periodic rupture of these vesicles.

All the symptoms of Meniere's Disease can be explained as a result of the contamination of the endolymph by this vesicular fluid. The violent vertigo is due to the hyperexcitability of the crista by the contaminated endolymph and not to the increase in fluid pressure.

The distention observed histologically in the endolymphatic system is the result of excess pressure from the vesiculation and toxic fluid production. This distention occurs most prominently in Reissner's Membrane because this is the thinnest part of the endolymphatic system.

The pressure of the contaminated endolymph takes the course of least resistance and therefore distends the cochlear duct and bulges or ruptures the delicate Reissner's Membrane.

The cumulative loss of hearing during the course of the disease is due to progressive damage of the Organ of Corti by the repeated contamination of the endolymph.

The tinnitus is the result of stimulation of the Organ of Corti by the contaminated endolymph.

The deafness that occurs during the attack represents a toxic loss of function in the hair cells of the Organ of Corti. These symptoms subside gradually because the contaminated endolymph is slowly resorbed.

In our explanation of the above symptoms we have denied any important role to the pressure.

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DISCUSSION OF THE THREE PRECEDING PAPERS

DR. SAMUEL ROSEN: It is obvious that there is no single cause for Meniere's disease. There are many causes for Ménière's disease, as there are many causes for practically every disease to which a human being is heir.

I should like to introduce a new structure which may have a great deal to do with Meniere's disease, a structure which has hitherto been believed to subserve only the function of taste, namely the chorda tympani nerve.

Recently Costen,Clare, and Bishop and we also have demonstrated that the chorda tympani nerve is a mixed sensory nerve and is not a nerve of pure special sensation at all.

The chorda tympani nerve, ends in the geniculate ganglion and goes upward as the nervus intermedius to end in the sensory 7th nucleus. The nervus intermedius, connects with the vestibular branch of the 8th nerve. The intermedius then goes centrally to end in the fasciculus solitarius along with the 7th and 9th nerves. Impulses that arise in the chorda tympani are carried up the nervus intermedius which is in close association with the vestibular nerve peripherally and with its nuclei centrally. This pathway could explain the vertigo and tinnitus in Meniere's disease.

Operating upon several patients with Meniere's disease under local anesthesia through the external auditory canal was facilitated by lifting up the ear drum and exposing the chorda tympani nerve. I thus had an excellent opportunity to test the function of the chorda tympani by electrical stimulation.

This illustration (slide) shows the drum reflected up on itself and here you can see the chorda tympani nerve resting on a bipolar electrode. I stimulated the chorda tympani with the following results—

When I stimulated the chorda tympani nerve in 36 cases, I got vertigo in one, that is, just as soon as I touched the chorda tympani, the patient said, "I am going around and around just as I did when I get a severe attach." Two had vertigo and tinnitus. Since I made this slide I have had three more who have complained of vertigo, so that there are six cases that complained of vertigo during the stimulation of the chorda tympani. 31 had tinnitus alone. None of them had taste all 36 had pain and all 36 had facial contraction. I mention this because I believe it is novel to find that stimulating the chorda tympani nerve should give vertigo or tinnitus.

In cases of Meniere's disease I have found that in the vast majority of them merely sectioning the chorda tympani nerve and putting the drum back again has stopped the vertigo as completely as if the labyrinth had been destroyed. Since this is such a simple procedure it certainly should be done before a destructive operation on the labyrinth is performed.

There is one more observation that I would like to tell you about. I have two patients who had severe Meniere's disease, one over a period of three years, which followed the complete destruction of the labyrinth secondary to mumps on one side. The other was a patient who following fracture of the petrous pyramid, also had a dead labyrinth, followed by several months of severe attacks of Meniere's disease with falling and paroxysmal attacks of vertigo. Cutting the chorda tympani nerve in both of these cases has abolished the vertiginous attacks.

DR. HENRY L. WILLIAMS: Dr. Lempert's presentation has been of very great interest. I think that one weakness one might call attention to in his presentation is his exclusion of one of the more important hypotheses as to the genesis of Ménière's disease. Some 40 years ago Shambaugh suggested a hypothesis of vasomotor labyrinthine ischemia as the initial cause of Meniere's disease. This hypothesis was further elaborated by Lermoyez and since by a great many other observers of this condition.

Since vasomtor labyrinthine ischemia is an attack of autonomic dysfunctoin and since we know autonomic dysfunction effects are almost if not quite synonymous with what we call physical allergy, and since we know all allergies are associated with disorders of the peripheral vascular bed, whether an antigen-antibody complex is present or not, I think that it is fair to state that one of the principal hypotheses of Ménière's disease has been allergy, or, if you wish, autonomic dysfunction of localized type or physical allergy, which I think most people consider to be synonymous terms. Under this hypothesis one assumes the allergy, when it affects the stria vascularis produces an altered type of secretion of endolymph containing a higher concentration of protein with a higher osmotic pressure. This would tend to attract fluid from the perilymph space through the semipermeable membrane of the endolymphatic system. The fact it has not affected the vestibular membrane in the cases Dr. Lempert present might be explained on the hypothesis that the diffusion of this altered endolymphatic fluid of higher osmotic pressure had not proceeded far enough to involve the endolymphatic system further than the ductus cochlearis.

Dr. Lempert has unconsciously supported the hypothesis of allergy very strongly in his histologic sections. He shows the presence of collagen disease of the subepithelial tissue which is characteristic of allergy. It is well known that the first pathologic change which one sees in allergy is in the collagen. This has been demonstrated frequently by Klinge and other investigators. I believe instead of the very fanciful hypothesis, supported by anything but the evidence rising out of Dr. Lempert's very active cerebral convolutions which he has presented. His demonstration actually supports the hypothesis of allergic origin of Meniere's disease alone

I was also very much interested in the presentation of Drs. Lindsay and Schuhknecht. I think that as far as the ductus cochlearis is concerned there has been suggestive evidence that in the human being this duct is not open as in the laboratory animal but is closed by bone and fibrous tissue. I believe Meurman of Helsinki, who was with us last year, investigated this problem and felt in the human there was not a free communication between the subarachnoid space and the endolymph but rather it was a closed tract. The experiment in the cat in this instance probably is not of very marked significance as far as the physiology of the human labyrinth is concerned. As Dr. Lindsay said, the experiment of closing off the endolymphatic sac and duct may be of greater significance. The negative result might be explained on the basis of the fact that he was dealing with normal states of endolymph. It would be relatively easy for the excessive normal fluid to be carried off by transudation through the semipermeable membrane of the endolymph system into the perilymph or escape into the blood in other ways. On the other hand, if pathologic conditions are present in the endolymphatic sac and especially the perilymph a functioning otic sac might be of more importance. We ought not to forget that the endolymph is actually lymph or in other words essentially a collection of interstitial fluid.

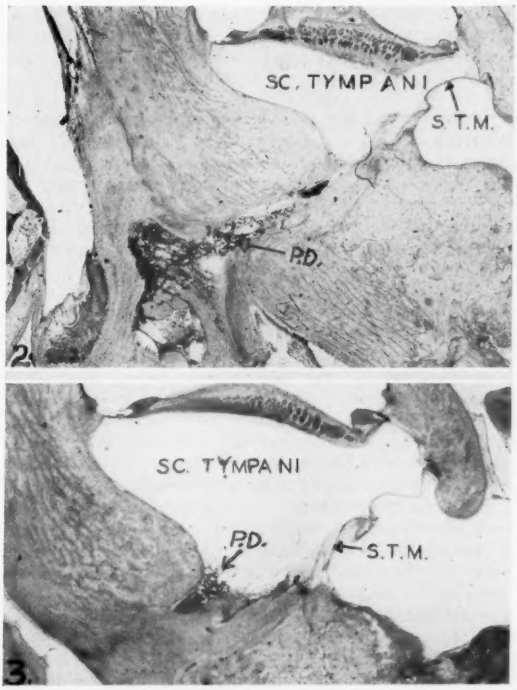
I feel the important factor that Dr. Furstenberg brought out in his consideration of the nervous anatomy of the vestibular system was the tremendous im-

Fig. 1.—Photograph of the posterior cranial fossa of a 62 year old man showing the large right and left otic sacs (o.s.) which were injected with india ink and seen through the layer of the dura. At the point marked (X) the sac dips under the shelf of bone to continue as the otic duct at the mouth of the vestibular aqueduct.

portance of differential diagnosis in Ménière's disease. I think that it is unfortunate that some individuals introduce in their discussions the term "Ménière's syndrome" or "symptom complex" because that is very confusing terminology. Almost any condition associated with vertigo, nausea and vomiting, whatever its origin, is classified as Ménière's syndrome. If we continue to use this terminology I am sure that we will not infrequently miss the diagnoses, as Dr. Furstenberg so ably indicated in his review of the anatomical factors involved in various types of vertigo nausea and vomiting which are not Ménière's disease. He also drew attention to those signs and symptoms which ought to alert us to the fact that some condition other than Ménière's disease is present. Ménière's disease, according to my conception of the condition is an allergy usually of a physical type but occasionally of the antigen-antibody type which may involve the stria vascularis, producing cochlear symptoms; may involve the aterioles supplying the cristae and macula; or may involve both together. Thus on an anatomic basis we may have the usual form of Ménière's disease with both equilibrial and cochli or symptoms or Ménière's disease without vertigo (endolymphatic hydrops save vertigo) or Ménière's disease without deafness (or without endolymphatic hydrops). Almoth Wright found that these last two varieties compose about ten per cent of cases of Ménière's disease.

T. H. Bast, M.D.: Mr. Chairman, Members of the American Otological Society and Guests. First of all, let me congratulate Dr. Lindsay on a very fine presentation of the results of carefully planned and expertly executed researches by himself and Dr. Schuknecht on the otic and periotic ducts. I had the pleasure, several years ago, to study some of their preparations and can substantiate the accuracy of the essayist's report.

Two years ago at the Society's Meetings at San Francisco, I presented evidence for Dr. Anson and myself that the human otic sac was very large, was surrounded by a rich vascular plexus which drained into the sigmoid sinus, and that the



Figs. 2 and 3.—Photomicrographs at two levels through the periotic duct of a cat. In this cat 1 cc of hydrokollog solution, injected into the cysterna magna, slowly replaced 1 cc of cerebrospinal fluid without detectable change in pressure. After twenty minutes the cat was sacrificed. The carbon pigment is seen in all parts of the periotic duct (P.D.). In Figure 2 the periotic duct is cut at level where it communicates with the subarachnoid around the glossopharyngeal nerve and ganglion. In Figure 3 the periotic duct opening into the scala tympani is filled with the carbon pigment.

P.D.—Periotic duct. S.T.M.—Secondary Tympanic Membrane.

epithelium showed characteristics found in other epithelia known to have resorptive powers. This all seemed to add up to the older interpretations that the otic duct and sac served as a drainage place for endolymph. Figure 1 is a photograph of the posterior cranial fossa of a 62 year old man in which the right and left otic sacs (o.s. Fig. 1) were injected with india ink to show their patency and large size. The otic sac is a prominent structure measuring about 12 mm in length. This is a large structure when compared with the rest of the internal ear whose length is only 17 mm.

The essayist obliterated the otic duct in the cat by entering the hiatus of the aquaeduct vestibuli—(at the point marked X in Figure 1) thus interrupting the continuity of the sac and the rest of the otic labyrinth. Up to the time of sacrifice of the animal no structural or functional changes were noted. In other words as far as function of the ear is concerned this large prominent part of the otic labyrinth, the otic sac seems to be of no significance. Here then are two sets of observations the results of which are diamtrically opposed. What shall we believe?

The second structure under fire is the periotic duct. Figure 2 is a photomicrograph of a section through the periotic duct, of a cat, where it communicates with the subarachnoid spaces around the glossopharyngeal ganglion. (Note carbon pigment in the duct and the subarachnoid around glossopharyngeal nerve.) Figure 3 shows the same periotic duct as it enters the scala tympani. (Again note carbon pigment.) These sections were obtained from one of the animals used by Dr. Settlage, from my department, in his studies on cerebrospinal fluid and its pressure changes. In this cat one (1) cc of cerebrospinal fluid was withdrawn from the cisterna magna so slowly that there was no noticeable pressure change, and immediately replaced by one (1) cc of a solution of hydrokollog. After 20 minutes the cat was sacrificed. Without apparent change in pressure, the hydrokollog passed through the periotic duct into the scala tympani as shown in these figures. In the cat, therefore, there can be no question that there is free communication between the scala tympani and the subarachnoid spaces. Now, today we were shown evidence that this periotic duct in the cat can be destroyed without altering the functioning of the ear. What shall we conclude?

In research we must consider all observable facts which often are contradictory. The dominating influence of preformed concepts have no place in research. When new conflicting observations appear it is the job of the scientist to sift and winnow all evidences. Truth alone will stand the test of time.

In evaluating the evidences presented by the essayist, at least three facts should be carefully studied.

1. Removal of an organ or part of an organ without apparent disturbance of function does not necessarily prove that the organ has no function. For example, one needs only to mention, destruction of parts of the periotic labyrinth as in fenestration, or removal of the spleen, the epiglottis and others.

2. In forming concepts about movements of either otic fluid or periotic fluid one must bear in mind that such movements are extremely slow, not perceptable movements as the term "flow" might suggest. To obtain functional disturbances from blockage of such slow fluid movements may take a long time, years or more.

3. In experimental procedures of this type, physiological conditions are created which differ from the normal. One of these is increased vascularity which

may persist for a long time and which may take care of any fluid balance which normally may, in part, be subserved by the structures removed.

These are only a few apparent factors which should be considered. There may be others. Knowing the scientific attitude and the splendid research program of Dr. Lindsay and his staff, I feel confident that all evidences will be considered and that forthcoming observations will lead us closer to an understanding of the function of this complicated organ—the ear.

DR. STACY R. GUILD: Dr. Furstenberg's presentation is extremely timely, in view of the tendency to call everything that has vertigo Ménière's disease. Pathologists realize the situation very well, as do also the members of this audience, but there are many internists and specialists in other branches of medicine who do not realize the facts Dr. Furstenberg has presented so ably.

Secondly, with regard to Dr. Lindsay's report: my first appearance before this Society, in 1927, was to report the studies to which reference was made by Dr. Lindsay. I am delighted to have checkups and further studies of the topic. For about 15 years after that report in 1927 I repeatedly urged various people in this country and abroad to attack the problem, not simply to accept the conclusions which I had reached. Nothing was done but to quote my studies in articles and in books. I am therefore especially delighted to have further evidence brought out. I am not willing to admit, however, that the answer is fully obtained as yet.

As to the function of the endolymphatic sac, I think that in the normal animal it has a resorptive function, as was indicated by my observations and by some other items. The experimental observation, I would like to point out, as done originally merely provided a clue to the function of the normal unoperated, uninterfered-with endolymphatic sac. This structure, not only in the guinea pig, but in the cat, in man, in the frog, in the reptile, and in other forms, contains its own evidence that the movement of the endolymphatic fluid is in that direction. The evidence is provided by the cellular debris that accumulates in the saccus. Nowhere else in the entire labyrinth does any collection of intact cells or cellular debris accumulate. I listened intently this morning for a statement by Dr. Lindsay as to where this material went in the animals kept for eight and nine months after the experimental operations. Where does this material accumulate that previously went to the sac? I hope that he will, if he does not have the answer ready this morning, tell us later whether an accumulation of such material has occurred and where?

With respect to the portion of the work which Dr. Schuknecht presented, on the cochlear aqueduct, I wish to add some observations. Dr. Bast also referred, as did one of the other discussers, to the matter of the human cochlear aqueduct being patent or not patent. I agree with Dr. Bast's statement that it is usually parent. It is very narrow, not wide like in the cat. Only in the human fetus and in the very young infant is the cochlear aqueduct wide. In the adult it is usually patent but narrow, and filled with this very densely packed arachnoid mesh material to which Dr. Bast referred. The evidence that not only histologically it appears patent but that it is functionally patent was reported to this Society at its meeting in 1928. Dr. Crowe and I presented observations at that time on material obtained from Dr. Dandy's neurosurgical service, in which postoperatively the red blood cells of the patient, not carbon granules, or something else, were in the scala tympani. The red blood cells could be followed, by inspection of serail sections, up through the cochlear aqueduct. That is evidence it was functionally patent.

Although usually patent, the cochlear aqueduct may be occluded in man. I have observed three such cases. All were in young adults who had had normal hearing. The histologic sections revealed completely obliterated cochlear aqueducts in the narrow part. This observation agrees perfectly with the experimental occlusions reported.

I would like to turn just for a moment to the report by Dr. Lempert and his associates. That is a perfectly fascinating approach to an unsolved problem. For many years I have been seeing in histologic prearations, as I think everyone has who studies human material, these thickenings, these bleb-like irregularities of the inner wall of the membranous semicircular canals. They are present so commonly that I agreed with the statement by Hallpike which was quoted by Dr. Lempert. It is an intriguing thought that these structures of common occurrence may have pathological significance. If the idea is correct, then I am certain that in this room today there are many cases of subclinical Ménière's disease.

DR. GEORGE SHAMBAUGH, JR.: Dr. Lindsay and Dr. Schuknecht are to be congratulated on their fine piece of work. I think that we should remember that the normal body has a large margin of physiologic reserve. For example, the stomach may be removed, and yet the patient continues to digest foods. From this, one might erroneously conclude that the stomach is not concerned with digestion. The fact that the endolymphatic duct and sac and the aqueductus cochlea can be removed and the ear remain normal does not necessarily mean that these structures do not have function.

Dr. Lempert's work is extremely important and provocative. We should keep in mind that the endolymphatic labyrinth is of ectodermal origin. There are three tissues in the human body which have developed to a very high degree the ability to produce antibodies. These are the skin, the respiratory mucosa, and the gastrointestinal mucosa. The reason is obvious: these are the usual portals of entry of organisms trying to enter the body. This ability to produce antibodies is possessed by the epithelium of the endolymphatic system, which is derived from the skin. The vesicular lesions observed by Dr. Lempert may be analogous to hives on the skin—the results of an allergic antigen-antibody reaction. There is no question but that some cases of endolymphatic hydrops that we see clinically can be traced to a specific allergy to a food or other foreign substances.

DR. BENJAMIN H. SHUSTER: I wish to limit myself not to Meniere's syndrome, but I would like to say a few words about the anatomy described by Dr. Furstenberg. I think it is about time that some clinician with a methodical mind like Dr. Furstenberg studies this anatomy to see if something can be done to clarify it. The textbooks on neuro-anatomy vary, in their description of the nerves, not only in their names but in their courses. The more Dr. Furstenberg read and the more I recalled some of the other pathways, the more confused I got and even some of the clinical applications of the anatomic points that he brought out were confusing. For instance, he takes up the pathways from the vestibular nuclei to the 6th nucleus and somehow or other crosses to the third on the opposite side. Bing a number of years ago described the innervation of the third ocular muscles as being such that a lot of these things could be explained. For instance, the third and sixth, nerves; those parts of the 3rd nucleus are usually bilaterally innervated. The levator palpebrae and the superior rectus have only unilateral innervation and the medial rectus as well as the inferior oblique have bilateral innervation because the muscles they supply have to act in unison with muscles of the opposite side.

The quick component of nystagmus has been described as being in the cerebrum. It has been described by Lorento de No as being in the reticular formation in the brain stem. Spiegel of Philadelphia said that this is wrong, that the impulse of the quick components is within the vestibular nuclei. I hope that Dr. Furstenberg finds the right one.

There is one term that I think he uses in which he refers to the rotatory nystagmus as irregular. I think that is an unjustified attack upon the poor rotatory nystagmus. Others have emphasized the horizontal component in the alternate hot and cold caloric test. They simply take the reaction or records of the horizontal canal and ignore the superior vertical canals. It is quite important to know whether the vertical canals react normally or not, or react at all; or whether there is a quick component or not. I do not think we can dissociate these facts and say one is regular and the other is irregular.

Another point in Dr. Furstenberg's description is the connection of the lateral nucleus and the medial nucleus with the horizontal canal only. I believe he said that they control the function of the horizontal canal. We know that in seasickness, when a person is in a position in which the horizontal canal is mostly stimulated, seasickness is not as great as when he is put in the position where the vertical canal is stimulated. It has always been my impression that the medial nucleus is associated with cells in the vegetative system in the reticular formation which causes nausea and vomiting. Many speak of seasickness as vertigo. There is no vertigo in seasickness. Most of the reaction is a vegetative reaction of pallor, sweating, etc. and that has nothing to do with vertigo.

One point more is surprising and that is the brevity of the reference in this discussion to any motion. There is only a statement that the vestibular nuclei are connected with the cerebellum. Vertigo is a disturbance of the sense of motion. It may originate in the temporal lobe more than other lobes but the cerebellum is deeply concerned with co-ordination and any disturbance of co-ordination is a disturbance of the sense of equilibrium. It is interesting to note the complexity and extent of motor impulses of equilibrium which when disturbed causes vertigo. These are propioceptive impulses from joints and tendons which travel up the same side. Then from the opposite side come the cortico-pontine cerebellar pathways to the cerebellum. There are no direct exits from the cerebellum to the cord. Before such an exit takes place, the cerebellum calls upon other connections through the red nucleus in the midbrain on the opposite side. In the red nucleus are connections with the optic thalamus, basal ganglia for fine automatic movements, and the cortex of the temporal and frontal lobes. The cerebellum sends the superior cerebellar peduncles to this red nucleus on the opposite side, brings all those impulses back to its own side as the rubro-spinal tract which goes out of the spinal cord with the pyramidal tract in the motor pathways.

This is in keeping with Dr. Fursteneberg's plea and the emphasis by Dr. Williams that all vertigo is not necessarily labyrinthine. These mentioned pathways may be involved in multiple sclerosis, tumors, vascular disturbances, notably thrombosis of the posterior-inferior cerebellar artery. This latter condition is easily confused with Ménière's Disease. This includes the effect on other areas pointed out by Dr. Williams such as the innervation of the tongue and the pharynx. We otologists should have some acquaintance with neurological facts which concern our specialty.

DR. ALBERT C. FURSTENBERG: I am very happy to have Dr. Shuster discuss the neuro-anatomy of this region. I think he has brought out some exceedingly important points.

As you can well understand, this is a very complicated region in the vicinity of the 4th ventricle; the pons and medulla and some of these diagrams are complicated enough to make it difficult to describe the anatomical pathways from the pictures presented here on the screen.

I should say, Dr. Shuster, that in sending the vestibular impulses to the vestibular nuclei; then across and up to the abducens nuclei, and then over to the oculomotor nuclei, that this was done only for academic purposes. I wanted to show that the contralateral medial rectus was being stimulated by this innervation and it seemed to me that in this diagram that was the best way to handle the situation.

Finally, let me say that I am primarily a clinician. I am interested in these anatomical pathways and I think we must review them from time to time if we are to understand our clinical observations. Over the years we have had many patients referred from the Department of Neurology who have complained of the typical vertigo of Meniere's disease, as well as a hearing loss, nausea and vomiting. Some of these patients have had excessive lacrimation on one side, flushing of the face, burning in the throat, rapid respirations, numbness of the ear and face, perhaps a typical tic douloureux. Don't make a diagnosis of Meniere's disease in these individuals and operate on the labyrinth. Give yourself a little time and demonstrate that there are perhaps other neurological conditions responsible for these symptoms.

DR. JOHN R. LINDSAY: First, I think Dr. Guild and Dr. Bast have given the answer to Dr. Williams' comment on the patency of the cochlear aqueduct and the periotic duct in the human.

I want to thank Dr. Bast and Dr. Guild particularly for their comments.

I am anxious that nobody gets the impression from our presentation that we would presume to deny that the endolymphatic sac and duct have any function. Such, of course, is not proven by these experiments. We called attention to that in the paper. The experiments do seem to prove that the labyrinth can get along without these structures, without having demonstrable functional changes or histologic changes. We have been particularly interested in that fact since it is evidence that disease of these structures is at least not concerned with the etiology of Meniere's disease, or possibly other diseases of the labyrinth.

Regarding Dr. Bast's comment on the movement of the fluids being extremely slow and that it might take a long time for changes to make themselves apparent, I think we are aware of the function which the endolymph must perform. The sense organs are suspended in the endolymph and must depend on the endolymph for their oxygen supply and nutrition. The experiments on anoxia are illuminating because they show that in a very short time the function can be knocked out, as has been demonstrated by Wever and Lawrence. Therefore the endolymph must perform a very active function at all times.

As to Dr. Guild's question as to where this accumulation of debris ordinarily seen in the endolymphatic sac goes, we have not been able to find it. We have not seen it so far, which of course raises the question as to where it came from in the first place. Did it come down through the duct or did it by some means or another arise in the sac itself?

LXV

NOISE IN INDUSTRY; MEDICOLEGAL IMPLICATIONS

C. Stewart Nash, M.D.

Rochester, N. Y.

Dr. Nash read a paper with the above title which was previously delivered at an inservice training course at the School of Public Health at the University of Michigan on February 8, 1952, and printed in *Industrial Medicine and Surgery*, in April 1952.

DISCUSSION

Dr. Howard P. House: The experiences of Dr. Nash in the industrial noise field are second to none and he has made many invaluable contributions to our knowledge of this subject.

In 1947, the American Academy of Ophthalmology and Otolaryngology, through the Conservation of Hearing Committee, appointed a Subcommittee on Industrial Noise. Dr. William Grove was appointed its first Chairman and through his activities funds were provided from several private sources, as well as through the Academy to employ a full-time research man in the field to study the industrial noise problem. He was fortunate in securing the services of Douglas E. Wheeler, Ph. D., who is still actively carrying on this work in numerous plants and industries throughout the country. We hope through his activities to obtain the answers to some of the problems which are concerning all of us. Such problems include the intensity levels which may be damaging to the human ear, the frequencies which seem to be providing most of the damaging effect, and the problem of individual susceptibility. He is carrying on research at the present time pertaining to a susceptibility test which may allow us to diffrentiate between the susceptible and non-susceptible individual. The problem of standardization of calculating hearing loss is most important in these cases and needs agreement and clarification. The matter of permanency of hearing loss and the relationship of the temporary threshold shift to this problem is of grave concern.

Until some of these perplexing questions can be answered we must encourage our sound engineers and architects to design machinery and factories so as to minimize the problem of noise. Likewise, much can be done, and is being done, with regard to the development of a satisfactory ear protector, which will be acceptable and worn by employees working in high noise levels.

Every plant having a noise problem should be routinely doing audiometric hearing testing on all pre-employment persons entering high noise levels, as well as periodic testing of those individuals following their employment.

DR. PERCY E. IRELAND: I am very much intrigued by the subject of Dr. Nash's paper. It seems to me there is a lot of emphasis on the medico-legal aspect of this problem.

We have been interested here in Canada, in conjunction with the Compensation Boards in the Provinces of Quebec and Ontario, in this problem. We reported to them we had made a survey in a monument plant outside of Toronto and in a screw and gear plant which is situated in Toronto and very noisy. I think Dr. House stole a good bit of what we felt should be done with regard to using ear wardens and things of that sort and I do feel that we have got to be careful in not getting beyond our depths. In fact, the man we have, Mr. Hodge, and the various other Ph.D.'s and not M.D.'s are the only ones entitled to figure out the acoustics which are a problem in this.

Our relationship with the Workmen's Compensation Board is very pleasant and also with the labor unions in this regard. I am very much alarmed by the report of all the disputed cases that are coming from the State of New York. Our Workmen's Compensation Board does recognize that there is noise in industry and will pay compensation for it. I think that the labor unions realize there are a certain group of people who have good jobs and don't care whether they hear or do not hear. They are not going to work at the menial job of sweeping a floor when they get $3.00 an hour for a job where they are trained in making gears. It is very much like the early days of compensation for silicosis. There was very much objection to the question of silicosis when it came through first, especially in our mines in this country, where the workman, if his chest plate showed silicosis, must work above ground and his income was reduced greatly. The labor unions are willing to play ball. They say "We will start from a certain point and you test us and if we have got damage to our hearing, that is all gone and past and let us from now on do something for the other fellow."

I think most of this is a matter of education. I believe the labor unions are very much interested in just what Dr. House said, that is, what can we do to help this noise in industry and what can we do in the way of ear wardens? What can we do to quiet our job down? They are very much interested in that side of it. When it comes to the question of compensation, are we going to pay for mental anguish, heart balm, or what actually keeps them from earning a living? Compensation isn't insurance. Compensation is compensation for inability to earn normal money in their civilian life as compared with what they were earning before. I think there has to be very careful consideration of just what constitutes compensation, whether it is just a payment for the fact that they cannot hear well over the telephone or compensation for being unable to do their job as efficiently due to the damage that they have had.

DR. DOUGLAS MACFARLAN: I would like to ask one question of Dr. Nash. I think it is rather an important one. The court asks you, "What is the amount of noise that is dangerous or damaging?" I rather suggest that we avoid setting any percentage as to what is the damaging decibel noise level, because as Dr. Ireland has stated, there is a difference in susceptibility to noise damage. We must avoid in court and elsewhere, particularly in our committee reports, any statements that such and such a percentage of noise is or is not liable to cause the deafness. I wish that Dr. Nash would develop that point a little and tell us if any of the commissions of otologists have considered it.

DR. JACOB S. WENDEL: If Dr. Nash has time I would like to have him tell us a little about the pathology. He differentiates between traumatic and industrial deafness. It is not quite clear to my mind as to what the differentiation is.

If we are going to take up this question the way it should be taken up, we should have to know what the hearing of the men was before they began work or when they started to work, and I would like to ask what he considers an adequate hearing test before the men start to work, whether it is a test made by a nurse in the industrial plant or whether it has to be done by specialists.

Also the question of the protectors for the ears, I am wondering just what the status of that is at the present time.

I would like also to ask what is the relationship of the American Medical Association Committe to this work.

DR. C. STEWART NASH: Dr. House is my boss and he and the Committee on Noise in Industry are a great consolation to me because they give me medical and moral support in a fight that is rugged. Believe me, it is rugged.

Dr. Ireland says that the emphasis of this problem is on its medico-legal aspects. I agree with him. As a matter of fact there was no problem at all until its medico-legal aspect developed. In other words, men have worked under conditions of industrial noise for many years, some of them for 30 years or more and they were perfectly contented until labor leaders informed them that here was a compensable disability.

I spoke to Mr. Scott Stevenson and to others about the pay-as-you-go compensation that men in hazardous employment, receive in the form of increased wages. In Buffalo there are men in a shop who earn $200 a week, because they are skilled and productive drop forge hammer operators but they are no more skilled, than the men in the supply room or the men in the store room. The drop hammer operators earn $200 a week, but the other men earn $60 a week. It seems to me that the difference between $200 and $60 or $140 a week, which is a goodly sum, represents the pay-as-you-go compensation for the hazard that is present in the one case, but is not present in the other case. The law says no.

Dr. Ireland mentioned silicosis. It is my impression that in the State of New York the compromise so far as the industrial deafness is concerned will be exactly the compromise brought forth in the handling of silicosis.

Dr. Macfarlan raises a question which is unsettled, a question which is exceedingly important and a question which is causing a tremendous amount of study at this time, namely, what is the critical noise level? I do think we ought to make a definite statement about the critical noise level at this time because there is too much controversy. I might say to Dr. Marfarlan, that the noise level in the ordinary Pullman car is about 90 db and ask him if he ever heard of a Pullman porter having occupational deafness?

Dr. Wendel would like to know something about the pathology of industrial deafness. So would I. About one month ago I testified in court, and ran up against an attorney who had a different approach to occupational deafness. His questioning ran like this—and this is appropos of Dr. Wendel's question. He said, "Doctor, exactly where is the lesion located that produces occupational deafness? What is the lesion of industrial deafness? In what part of the ear is it located?"

I said, "Well, it is in the acoustic nerve tract."

"Where is that?"

"Probably in the organ of Corti."

"How many cells are there in the organ of Corti and how many rows are there in the organ of Corti? Doctor, would you tell me please exactly what an acoustic nerve cell looks like?" Continuing he asked, "If an acoustic cell is fatigued, what does it look like? If this cell has permanent damage, like an occupational deafness, what does it look like? If it has occupational deafness with acoustic fatigue, what does it look like, and how does it change when the acustic fatigue is removed? Add presbykousis to that, what does it look like? How does the acoustic nerve cell compare with the sciatic nerve?"

I said, "Well, so far as I know there are at present no definite answer to these questions. I don't think anyone knows definitely what these cells look like under the various conditions you mention and we are not absolutely sure that the cells are changed anatomically but they may be changed functionally or physiologically."

He said, "Doctor, have any autopsies been done on the people with occupational deafness. Has there been research done on occupational deafness ears?"

My answer was, "Not sufficiently to answer your questions."

His reply was, "Really you don't know very much about occupational deafness do you?"

Of course, the answer to this type of questioning is that a man may have epilepsy all his life and may have a perfectly normal brain macroscopically and microscopically or a man may be a raving maniac and be committed for 20 years and still have a perfectly normal brain and perfectly normal brain cells.

LXVI

ESSENTIALS FOR AUDITORY REHABILITATION

FREDERICK T. HILL, M.D.

AND

ELIZABETH O. KOONS, M.A.

WATERVILLE, MAINE

This paper makes no pretense of being scientific. In fact it might be labelled pure propaganda. On that score, however, no apologies are offered, for propaganda, specifically directed in behalf of a worthy cause would seem justified. That, in this instance it may be indicated, would seem bourne out by the oft-heard statement that one would like to start some sort of project for the hard of hearing, usually in the form of a clinic for children, but, was awaiting the raising of some stated amount of money, always in terms of six figures. Certainly a desire for the ideal is most commendable and one should always keep his sights high, but not at the risk of depriving handicapped persons of needed help. This laudable desire for the ultimate may serve, perhaps unconsciously, as an excuse for doing nothing. But when this results in depriving unfortunate youngsters, afflicted with deafness, of their opportunity to gain this most important means of communication with their fellowmen, it rightly may be assumed to be inertia, masquarading as idealism.

Surely the ideal program of aural rehabilitation is an extensive one and calls for rather an elaborate personnel, equipment, and adequate financial backing. Walsh, in an excellent paper, presented before the American Academy of Ophthalmology and Otolaryngology, described such a program. With the extensive facilities of the Central Institute for the Deaf and of Washington University at his disposal, one could easily agree that such a program would be indeed ideal. We are all familiar with similar programs elsewhere in the country, usually centered in the university medical schools.

From Thayer Hospital.

Walsh described the function of a University Audiology organization as three-fold: namely, teaching, research, and service for the deaf and hard of hearing. He further stated that the "personnel must comprise an otologist, a physiologist, an anatomist, a physicist, a psychologist, experts in electronics, educators, audiometrists, and so on." And I'm sure he meant to include social workers.

While Walsh definitely states that this is a university organization of broad scope; unfortunately, the very extent of such a program tends to engender a feeling that little or nothing could be accomplished with more modest facilities such as might be available elsewhere than in a university center. While this was, we are sure, the farthest from Walsh's thoughts, we have a feeling it has been at times a determent, preventing the development of services for the deaf and hard of hearing where they were urgently needed.

With the remarkable growth of our country, especially during the last few decades, we, as a people, perhaps unconsciously have been prone to place more and more emphasis on bigness. We speak in admiration of the tallest building, the greatest bank, or the largest corporation. Size has become almost a fetish, to be worshipped and to be emulated. We are prone to forget our earlier pioneers who did much with little, who made what was available do, and do fairly well. Of course, one would like to have completeness in any project, with all the equipment and personnel, and with the funds to assure success. But to insist upon completeness, and to postpone action until something approaching completeness is at hand, oftentimes indicates a lack of initiative and of imagination.

It is our feeling that this emphasis upon the ideal program, in many instances, has prevented the institution of needed services to the deaf and hard of hearing. And as rehabilitation for the deafened is most effective in the childhood years this means that much valuable time is irrevocably lost. For these youngsters are growing up all the time. They cannot wait for the fruition of the ideal.

James Garfield once said that his idea of a college was Mark Hopkins on one end of a log and a student upon the other. This probably would not appeal to many college presidents today in their unremitting quest for added funds for expanding programs but it might have certain applications to our problem. The figurative log, with a teacher of speech reading on one end and a deaf child on

the other, certainly would be preferable to the "do-nothing" policy, too prevalent in vast sections of our country. And it's surprising how many youngsters could be accomodated upon that log and how many could be benefitted thereby.

Deafness, as we know, when once established is, for the most part, irreversible. Our efforts to combat this condition are limited largely to prevention, circumvention, (fenestration), amplification, and education. Otology has made remarkable progress in surgical measures in those cases where fenestration may be indicated. It is gradually adopting a more logical approach to preventative therapy, with broader conception of etiological factors, in contrast to the former concentration upon nasopharyngeal lymphoid tissue, important as this frequently may be. It is taking a more active interest, long overdue, in the possibilities of amplification which should result in more intelligent use of hearing aids, with less exploitation of the deafened patients. But, except in certain centers, it has shown little interest in the field of education, in the training, which in the severely afflicted, especially in the younger age group, presents the most effective means of rehabilitation.

It has been estimated that there are in this country some two million children with impaired hearing. In nearly half of the States provision is made for the testing of hearing of all school children but one may doubt the thoroughness and accuracy of the tests in many cases. Of even greater conjecture is the frequency with which anything remedial is done as a result of these tests. Our State of Maine for years has had a law requiring the annual testing of hearing in all school children but, for the most part, it has stopped right there. Simply passing a law, or making a regulation is not enough. And this problem cannot be passed on to the public health agencies, at least, in most circumstances.

We have these centers, previously referred to, where in addition to training and research, adequate service to the deaf and hard of hearing is provided for those so situated as to have ready access to these facilities. But not all of these handicapped youngsters are so located and more extended efforts to provide these needed services would seem indicated, even if this means utilizing a more modest program. So rather than insisting upon the ideal and waiting for it to materialize, why not try to do with what we have?

The important thing is what we can offer in service—not in facilities. What then might be considered as absolute essentials for such a program, not the ideal but the minimum, which might provide means to allow these handicapped youngsters to take their rightful place in society?

The first essential is an otologist, or an otological clinic where cases picked up in the schools, or by the public health nurses, can be examined and screened, and indicated measures prescribed. With rare exceptions this should present no problem either in personnel or equipment. The most essential component, of course, is a competent teacher. And here, unfortunately, may be the real and perhaps the only problem. For these are none too plentiful and may be hard to find. But one never knows what latent talent there may be in any community until a search is made. And once a person has been exposed to the satisfaction, the sense of giving, inherent in teaching, the deafened, she never becomes entirely immune. Even those now married and bringing up children of their own are usually eager to aid with their skills, at least on a "part-time basis."

And for our figurative log, what could be more appropriate than a hospital which always should be willing to provide space for such a worthy cause. Volunteer workers to assist should present no problem if the need has been made known. Too few people know what teaching the deaf child means. When they understand what his educational problems are, and when they learn what can be done for him and how it is done, they are invariably drawn to the work. We have found that by including educating the public in our program, our own list of volunteer workers now exceeds our demand. To be sure certain equipment is desirable but once the project is under way it's surprising how easily this may be acquired. People will contribute to a going concern when they would hesitate about a visionary project. And there is a great appeal to this work for the deafened.

In support of what we have just said may we describe our own experiences at the Thayer Hospital, not that our accomplishments are at all remarkable but to show what actually may be done "on the proverbial shoestring."

For 20 odd years we had seen the need for a program of rehabilitation for these handicapped youngsters. We have had the sad experience, many times, of having to tell parents that their child was

irreversibly deafened and that all they could do was to try to get him into the overcrowded State School for the Deaf when he was five or six years of age; thereby losing the most valuable years for training, from three on. Finally we developed enough initiative to try to do something ourselves. Searching for talent brought forth a former teacher in the Lexington School, now married to one of our college professors. For the first year we set up the project under the vocational education program of the State Department of Education, receiving from them a small stipend for the instructor. At the end of the year the Department of Education discontinued their sponsorship but, through the co-operation of the teacher, we went along without any financial support whatsoever. Later the project was incorporated in the program of the State Department of Health and Welfare where it has since remained. This allows a small stipend for the teacher and has proven entirely satisfactory.

We now have 31 youngsters in our classes, coming from all over the state. The Tracy Clinic technique, with the mother coming with the child, is followed. This is a necessity, for our plan is to have the children go to their local schools with their own age groups. Of course, this requires extra work on the part of the mothers. They are present at each lesson, see what is done in the class, and then repeat the work in daily lessons at home.

At the clinic we start the three or four-year old with the fundamentals of speech and speech reading and gradually, as his comprehension of these increases and his vocabulary grows, we include "subject-matter," silent reading, number concepts, and writing. By the time he is old enough to enter the sub-primary class, he knows as much as, and sometimes a bit more than his contemporaries. We try to keep him a little ahead of his classmates through the second grade. Experience has taught us, however, that by the time he has gotten successfully through the second and into the third grade, he wants to be on his own. After that he comes to us once a month for a check-up and for suggestions for practice in speech and school subjects as his needs demand. We have gradually acquired needed teaching equipment, including a "Trainear," hearing aids for those who could be benefitted by them, through unsolicited gifts from persons who have learned of our work. Consultants in pediatrics, psychiatry, etc., are available from the hospital staff. Both press and radio have been most co-operative in telling our story to the

public. So now our shoe-string seems to be growing more durable year by year, rather than wearing out.

We have purposely confined our remarks to the rehabilitation phase of the problem. All of the patients referred have been carefully screened either in the ear clinics or our office where careful audiometric studies have been carried out.

The clinic has had one unexpected and yet extremely valuable by-product stemming from the use of volunteer workers from the Women's Division of Colby College. These young ladies become greatly interested in the work and certain of them have planned to make it their career. We give these volunteers a series of lectures designed to acquaint them with the fundamentals of the problem, which has proven of value in cementing their interest.

All of this is mentioned, as before stated, merely to show what can be done with very little. Our program is indeed a modest one and we lack much in the way of personnel and of financial backing but we are offering services to handicapped children that they could not otherwise obtain. And the dividends realized in the progress made by these youngsters, are indeed most satisfying.

Our plea is not to insist upon the ideal, not to wait for the day when all desired funds are available, but to do with what you have, thinking in terms of service rather than of facilities. If you can find a skilled teacher for the role of Mark Hopkins, your log will soon become a source of pride and joy to you and an immeasurable boon to many unfortunate youngsters.

DISCUSSION

DR. HARRIS P. MOSHER: To me a very enjoyable and very human paper, and that cannot be said of all the papers I have listened to in other societies at other times. I am not mentioning this Society because I have not listened to all the papers.

To me this is a very appealing subject but for many years it has not appealed to the otologists. I won't say the harsh things I might say as to why it has not appealed to the general otologist, but it is only of late, in the last ten years that it has appealed.

I should like to say about three sentences on the word "shoestring." Dr. Hill chose the word "shoestring." My first sentence is a practical one, if you are practical and this is the sentence: if you break one shoestring, change both.

Now coming to the other thing on shoestrings, a shoestring often will accomplish much given the right man back of it at the right moment, which applies to the house as well as to the office. The shoestring has accomplished in business and in the arts some of our most successful enterprises.

To my mind, given the right shoestring you do not need a grant or a fund. You see the gentleman does not need either of them.

DR. FRANCIS L. LEDERER: I think Dr. Hill has been very modest when he tells us about "this shoestring" that now actually has both strings and shoes. I would like to use the aphorism of the American Hearing Society, namely that "Hearing is Everybody's Business." Actually Dr. Hill and his associate, Mrs. Koons, have demonstrated in principle, that hearing is more than a one-man job. It takes a lot of people in a group to provide total patient care.

We are also to be made aware of the fact that we are witnessing a steady broadening of horizons of public health and community welfare. No more are we interested in the purely microbiological effects of disease, but we have now become accustomed to concern ourselves with the socio-economic aspects of the patient's future, with the doctor taking a leading part in communal welfare. We have made tremendous progress in this country in public health. That is going on pretty well. But we are lacking in effort on the preventive side of medicine. Good otological practice is synonymous with good preventive practice. These cases with hearing deficits must be found. They must be first ferreted out in mass surveys. They must have thorough physical examinations and adequate treatment (both medical and surgical) and they must have a distinctive care relative to their rehabilitation (both physically and emotionally). They are to be salvaged, as it were, and Dr. Hill has chosen well the case finding in children because they are the future adults who lead a frustrated existence and become problems either to themselves or to their community. They must be prepared to take their places in society.

We realize that a small town may be just as productive as a large city. It does not have to be the ivory tower that is going to furnish a solution. Dr. Hill has exemplified the effort that should be going on all over the country. It is terrifying for many practitioners to come into a large institution and see complex equipment, interpreting research equipment as representing the minimum standard for everyday use. I think those authors who have been identified with writing papers reporting results of complex test procedures, have been somewhat traumatic and prevented practicing otologists from going on and doing a job, each in his own way, as Dr. Hill has demonstrated.

Hearing is the concern of many. While there is a distinct advantage in centralization of the effort in a small community, it can bring it to the level of the special needs of the community, so that cases that demand medical care can be treated adequately but the doctor must never lose sight of his position of leadership. While he does not have to carry out the activities of these ancillary groups, he must nevertheless know enough about the various facets of hearing loss and be concerned with the activities of the personnel, so that he can understand the subject at hand.

The university functions are much broader than at a community level. They are concerned, as Dr. Walsh has stated (quoted in Dr. Hill's paper), with education, with reasearch, but never should we allow ourselves as otologists to leave the patient and his goals in the background. He is our immediate concern. May

we also say, that for your enjoyment of further activities in otolaryngology, see audiology with its overall definition, including speech, because speech and hearing are inseparable communicative senses.

In the City of Chicago the University of Illinois functions only in so far as a guiding influence, not as a dominating force, so as to gather all those people and agencies together who have overlapping interests. All the organizations concerned with the education, welfare and health of the patient must be brought together, because this is not a one man job. In total care, no one group can go it alone.

It is important that you, as an otologist, get into the school, health and educational systems of the state, in providing information, guidance and care. We as physicians can assume leadership.

Informative booklets in Illinois (If You Have A Deaf Child and Deaf and Hard of Hearing Children in Illinois) have served to inform the population, because only through education can these objectives be achieved. An ignorant public will not be a very co-operative one.

At the comunity level, teaching the parents to understand their children and the basic difficulty is most important. They are, after all, the patients in the formative years and in the early stages of the treatment of the young child.

Attention is directed to our school system examination, using the pulsing audiometer for ferreting out cases of hearing deficits by means of mass surveys.

In the City of Chicago, we have come up with some interesting statistics on the basis of over 62,000 children examined during the past year. This was done under the direction of Dr. Richard Marcus and his group of trained personnel. A large percentage of these children (8 per cent) were found to have hearing deficits. Family doctors and otologists were alerted and there were follow-ups. The only difference between what Dr. Hill had in mind and our effort in Chicago was that this survey did not merely come up with figures. The otologists were contacted and definitive treatment prescribed. These children were examined by otologists not merely by lay or inexperienced persons.

So the overall picture was the finding of large numbers of children with remediable losses and others requiring medical care as the result of the group's recommendations. Of importance also is what it means to the child in regaining acuity. The City of Chicago feels that through this survey they are saving one million dollars a year through the salvaging of so-called educationally retarded cases. Seeing to it that these children are discovered early so that they do not lose grades, because a grade lost is a grade cost to a community.

Last but not least, since we have not discussed much of the adult hearing problem, I wish to mention merely this unholy subject of the commercial aspects of hearing aids. Certainly since we feel the importance of mental hygiene at the adult level, we are concerned with the type of education that a population is being supplied through the medium of advertising by these commercial sources. These adverse effects must be nullified by the very things that you would be doing for the adjustment to the hearing deficit. I call your attention only to the thought that these statemens made in the advertisements are vicious. They are contrary to all decent commercial representations and moreover provide an adverse form of mental hygiene.

MR. R. SCOTT STEVENSON: I feel rather diffident about standing up here, following Dr. Mosher and Dr. Lederer, because what I know of this subject I have learned here in the United States.

I happen, in our country, to be Chairman of the National Institute for the Deaf, which has been dealing with or trying to deal with this problem ever since before the first war, from about 1911. As Dr. Mosher states, it is only within the last few years that otologists in general have begun to take an interest in the rehabilitation of the deaf. I was looked upon as an accentric when about 25 years ago I began appearing in courts to try to get money back for some of these poor deaf old ladies who were robbed by the fellows who sold hearing aids to them. It was difficult—and no doubt you find it too—to persuade judges that it was not merely routine commercial practice. As a matter of fact, I gave up trying to get the money back after half a dozen or so cases because it appeared to be that the robbery that was going on might be immoral but still be legal. Anyhow our National Institute for the Deaf formed an approved list of hearing aid dealers, and, believe it or not, quite recently they have been scrambling to get on our approved list.

We laid down certain minimum ethical principles such as insisting on making no extravagent claims in advertising, in allowing free trial of a hearing aid before it is paid for finally and allowing no door to door canvassing, things of that kind. They are nothing very marvelous but they are at least minimum ethical principles and all but one or two of our hearing aid firms have accepted them. That is one thing we have done.

Another thing, as you found here no doubt, there is a shortage of teachers of lip reading. Lip reading, as Dr. Hill has said, is probably the greatest single aid in the rehabilitation of deaf persons, young or adult.

In our institute, when we found there were not enough lip reading teachers, we said, "All right, we will teach lip reading teachers." The first time we advertised to get people to teach lip reading, we got over 200 people wanting to learn to teach lip reading. We picked about 30 of them and we picked teachers to train them. In the last year we have about 80 persons, up and down the country, nearly all working part of the time, teaching lip reading to small groups. In fact, it has got to such a stage that the larger centers are quite adequately covered. We are now having to deal with the problem of the rural centers. It is more difficult.

I was particularly interested in one thing in Dr. Hill's paper. I preach very often that the foundations of modern otology after all were started in one room in Vienna by Politzer, with a curtain down the middle of it; a bed and operating theater on one side and an outpatient department on the other. If that is how the foundations of modern otology were laid, it is unnecessary to wait till you get such magnificent buildings as you have in the States in the Veterans Administration and the Central Institute for the Deaf. I admire them very much indeed, but I tell our people at home, "don't wait till you can raise funds like that. Start off with an otologist, someone who will fit the hearing aids and teach the lip reading." There are your minimum requirements. It is not necessary to do research. One gets the idea everyone has to do research but that is really unnecessary. What we really need to do is to do something for the deaf.

I was greatly heartened by hearing Dr. Hill's paper today because we in England have the idea that over here nobobdy can do any thing until they start an enormously extensive center. Five years ago we had four aural rehabilitation centers in our country. Now we have 48. Few of them are doing research. All are doing a good job for the deaf. Some are in small communities, some in university centers, but all are doing something. However, as Dr. Hill pointed out,

in doing useful work for much neglected people something as well worth while as reasearch is being carried out.

DR. THEODORE E. WALSH: I feel I have to say a word here because Dr. Hill quoted me somewhat wrongly. In the paper I gave in Chicago I was supposed to discuss the relation of the otologist to audiology. One had to bring in all phases of audiology in such a discussion but I think, at least I hope, that I made it quite clear that the main part of the physician's effort should be in service to the patient. It is true that in St. Louis we have the Central Institute for the Deaf started in Dr. Goldstein's office. It started with one man, in an ordinary office to which children came to be rehabilitated. It was the enthusiasm of one individual, one real otologist who built up this institution, although it is closely related I think Dr. Hill's message really is that an otologist, an individual, can do this job and does not have to wait for money if he puts himself in it.

DR. ARAM GLORIG: I would just like to say if there is any question in your mind about the worthwhileness of such a problem let me give you a few statistics from the Army. We have rehabilitated in our center approximately 5,000 men in the past three years. 98 per cent of these men have been returned to full military duty and are doing an adequate job. They vary from the rank of three star generals down to private. As a matter of fact, recently General Collins, Chief of Staff, has issued an order that all of the generals having hearing problems better get down to the Rehabilitation Clinic and get something done. So recently we have had the influx of a lot of rank.

Remember this too started on a shoestring. It began with Dr. Marion Mobley and a couple of lip reading teachers at Walter Reed back in 1943, and has since grown to be what you known the Army program is today.

DR. DAVID DE WEESE: I merely want to comment that I have been associated with this type of program in Portland, Oregon for the past six years. I wish to point out that when such a program starts in a small way, the most vociferous support will come from the parents of the deaf children. In Portland we had no lip reading teachers to begin the program. However, the parents of the hard of hearing children got together, formed a group and went to the school board. They raised the roof to the extent that the school board began giving local teachers a sabbatical leave with pay (with double pay now) if those teachers would take one year of training in special education for the hard of hearing and return to their teaching jobs in Portland. Parents also went out soliciting aid from the medical profession and were able to obtain additional funds from the local otolaryngologists to support the additional pay for these teachers. It was felt that local teachers feel more inclined to come back to the Portland area and continue teaching that if new teachers were hired from outside, even had they been available.

DR. FREDERICK T. HILL: I wish to thank all of you who so kindly participated in the discussion and have been interested in such an unscientific paper.

I was very much pleased that Dr. Mosher feels kindly towards "shoestrings," because I have had to work on one all my life.

Dr. Lederer brings out the importance of broadening the horizons in otology. That is very important. They tell us that if we are going to be good specialists we have got to be good physicians. I think we can carry it a step farther and say if we are going to be good physicians we have got to be good citizens. I am interested in his remarks about hearing aids. That has been a difficulty. Dr. Stevenson brought that out, too.

We have tried to develop a hearing aid center in our group for those children who could be helped by them, giving them an opportunity for trying out different hearing aids. As some of these young children come along under instruction we find by using the trainear that some of them can be benefitted by amplification. I was very much thrilled some time ago, when one of our older girls who had come to us with greatly impaired hearing and a terrible voice, after some training with Mrs. Koons and a well-fitting hearing aid, was announced as one of 25 qualifying for the finals out of a class of 125 people who participated in a public speaking contest in her local junior high school.

I was very pleased with what Dr. Stevenson said. It is quite consoling. I think we feel alike that we should start with what we have. His experiences with recruiting teachers is most encouraging. I think that we can interest these young people in this as a career and we have found that the college people are a very fertile field.

I did not deliver the written paper. I am very familiar with Dr. Walsh's paper. If he reads my paper I think he will find I quoted him correctly.

LXVII

PROBLEM OF HEARING DEFECTS IN CHILDREN

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It has long been recognized that more children can be protected from hearing loss by detection early in life, followed by treatment of the cause, than in any other way. In my opinion many present systems of detection are defective and treatment of the causes is almost non-existent.

I believe that the faults of detection are due to lack of critical ability on the part of the otologists who advocate or pursue the methods that are currently popular and the lack of treatment comes from widespread avoidance by otologists of a very difficult task.

A good reason for this state of affairs used to be the lack of adequate means of measuring hearing. The invention of the two kinds of audiometers brought about a change. The otologist, however, shrank from the responsibilities attached to the opportunities thus afforded. He handed over the detection to the school teacher and the the treatment to the ordinary physician: early hearing loss was discovered in school children by screening tests carried out in public schools all over the country: treatment for this was supposed to be carried out by private physicians and public clinics but has been notorious by neglect. And to-day there is a new bit of evasion by the otologist, which occurs in the fashionable new audiological clinics.

CRITICISMS OF AUDIOLOGICAL ESTABLISHMENTS

In many of these new establishments a considerable hospital distance separates the hearing suite from the otological clinic. To some of these, visits by any otologist are rare: the hearing establishment is run exclusively by the audiologist, his special assistants, his technicians, and his nurses: in the E.N.T. clinic proper there is no provision whatever for the testing of the hearing by the clinical otologist: the latter depends entirely on the reports sent from the audiological department.

The testing done in some of these departments is most elaborate and time-consuming—so much so, that in very few is there as much

as one attending otologist who is capable of performing the tests and no one who could afford the time involved.

Many new tests have been described and some of them strongly supported. Every audiological clinic has a formidable list of tests. As one noted critic states[1] "at least ten to twelve hours of time per patient would be needed to administer all the hearing tests now regarded by one or another investigator as of interest and value."

The assumption that these tests are of clinical value is not correct. Guild[1] has written three condemning paragraphs which have not been controverted.

"The information obtained from the elaborate tests or batteries of tests is primarily of research interest. Until the significance of the data obtainable by these tests has been evaluated in terms of lesions that otherwise would be overlooked or in terms of therapy that otherwise would not seem indicated, clinical otologists should not be urged to use them or made to feel that they are out of date if they do not."

"In other words it is time for a clear distinction to be made between tests for research purposes and those for clinical purposes. Nothing is gained clinically by the making of hearing tests that cannot be interpreted with reasonable certainly in terms of causative lesions."

"All too often a diagnosis, so called, amounts to nothing but a statement of the kinds of hearing defects found by the tests made. Such a statement, no matter how elaborate the battery of tests used or how detailed the report of the data obtained, is merely a description and not an interpretation of hearing tests and has no medical usefulness whatever."

The great establishments which I have criticised have a place in an institution devoted to research into problems of hearing and of psychology, but are unnecessary in a doctor's office or in a hospital clinic. In these there should be facilities which provide data that can be made use of for the good of the patient and the information of the otologist. A hospital clinic may have a research unit attached to it—but, as this unit cannot provide the information which the clinic and the otologist require, it is not necessary for hospitals to provide it unless the hospital can afford the extravagance of personnel for a research program.

The essentials of a satisfactory method of mechanically *measuring* the hearing are—accuracy, quickness, and a technique which can be exactly repeated at a later date. The apparatus should be as simple as possible, rugged, not likely to get out of order and constant in performance.

I believe that the facilities for testing the hearing should be in the E.N.T. suite and that they should be such that any of the attending staff can, when he desires, test the patient in whom he is interested. Further they should adjoin rooms where every type of clinical examination and treatment is available and both the testing and the treatment should be done by the otologist. I do not believe that the day of the otologist is over.

ARRANGEMENTS AT HOSPITAL FOR SICK CHILDREN, TORONTO

At our new Hospital for Sick Children in Toronto, the ear, nose and throat out-patient suite and the hearing clinic are housed together. Our out-patient clinic has been so designed that the entire suite of rooms is at the service of the hard-of-hearing clinic every afternoon. In addition, certain of the rooms are available to this clinic every morning. The clinic has been placed next door to the social service department and across the hall from the department of psychological medicine and the speech clinic. With all of these we work closely in the follow-up of our patients.

Because the history taking and the examination are of great importance they take considerable time to complete:—sometimes they are spread out over several visits. The child must find some features of its first visit pleasurable. The mother must sense that every effort to solve her difficulties will be made by sympathetic examiners of experience. Subsequent visits must confirm and enhance any happy impressions so that visits to the clinic will be looked forward to by both patient and parent. From the start the otologist must so conduct himself as to put the child at ease: because he wants the child to trustfully submit to whatever treatment or test he requires. Examples of disagreeable procedures which must never be omitted are—examination by nasopharyngoscope and the cold caloric test.

An account of the many stratagems which may have to be resorted to in order to achieve what has been outlined in the previous paragraph in itself would require a treatise.

Some of the little children we are asked to test have not developed powers of speech understanding and are without any demonstrable hearing. Some are infants in arms. Some cling to their mothers and others are completely terror stricken. Some are defective mentally. Some trust and obey the parent and eventually the doctor. Others remain frightful behaviour problems for a very long period in spite of every reasonable approach. Many such children fail to respond to auditory stimulation. However that does not necessarily indicate a hearing impairment. One child believed by a kindergarten teacher, by a social worker and by the relatives to have no hearing at all betrayed no response to any of our early efforts—and later, when he had been left alone for a period, was observed to obey his mother's commands given in a very quiet voice when he could not see his mother's lips.

ASCERTAINING HEARING IN VERY LITTLE CHILDREN

For the diagnosis of very little children who are extremely hard-of-hearing we have a number of devices: our own modification of the Dix-Hallpike peep-show, a melody master, numerous game-methods and a psycho-galvanic skin resistance apparatus.

Our peep-show was evolved from this thumb-nail sketch[2] after much experimentation. During the evolution of our model I visited Dr. Norton Canfield and adopted his excellent idea of centralizing all the controls on one board. It only requires one operator to handle our machine and the patient.

In this peep-show we can use either a coloured movie or coloured still pictures. We find that the movies captivate most of our little patients but occasionally a very timid child has to be tested with our set of still pictures.

We find the peep-show an invaluable first approach. The child can operate it without instruction. The picturesque little house at once disarms the child's fears and arouses his curiosity. He naturally looks into the doorway, but he sees nothing; for the black halway is in darkness. However, what is a door-bell-button for but to be pressed? The result is a pleasant discovery. A picture appears which is moving. The moving pictures of farm activities are pleasing and have dramatic attention-holding appeal. The desire to see what comes next keeps the child's co-operation. When his little arm is to weak to press the door-bell-button the patient can rest his arm on the ledge and press the door-step.

Over the doorway is a small red fanlight and behind the gable decoration is a loud-speaker. When the child presses the button a flashing light appears in the former and from the latter comes a sound synchronous with the flashing light. The light and the sound can be altered at will by the operator.

Four discrete frequencies—500, 1000, 2000 and 4000 cps are provided. The rate can be varied and also the relative lengths of the off and on periods.

In this test the child learns that when he responds to the flashing light by pressing the button he is rewarded by a continuation of the pleasing moving picture and also that when the light is not flashing, pressing the button produces no picture.

While this lesson is being learned the operator is experimenting with different intensities of the four available sounds. The control board can be operated without the child being aware of it. Sometimes the child will suddenly investigate the gable, thus betraying the fact that he has located the source of the sound. Sooner or later the operator switches off the light and in a favorable case the child continues to press the button whenever the sound is being produced.

The child has discovered that a certain peculiar sensation has a meaning; that if, when he perceives that sensation, he presses the button he will get the reward of more pictures. The pleasure the child gets is evident to all the observers and frequently brings tears to the eyes of the mother who up to that point has believed her child could not hear. The child has discovered that sound has a meaning and hope has been kindled in the mother's heart.

The doctor, on the other hand, has formed a rough idea of the degree and extent of the hearing loss by having observed the intensity of each note that has reached the child's intelligence. A very rough audiogram for four tones can be obtained in some cases.

This happy result can be the early stage of pure-tone testing for the little patient. If we had proceeded differently and had introduced the child to the pure-tone audiometer we would probably have aroused resentment and fear. For a child is not attracted by the uninteresting sounds of a pure-tone audiometer.

To keep the child's attention it is necessary that there be as few distractions as possible. Therefore, as a rule, no one else is in the

room except the child and the otologist. However, it is often necessary that the mother sit beside the child. Observers are accommodated in the observation gallery where they can hear and yet are not seen.

Our other devices for interesting children in sound will only receive brief mention.

When we have gained the child's interest and confidence by using the peep-show we endeavour to introduce him to head-phone hearing by using the Melody Master. The child is gradually persuaded to wear earphones through which he hears gramophone records of music. The mother assists by using a duplicate set of head-phones and by endeavouring to speak to the child through a microphone. The method is of only slight value at the initial visit but is very useful after the child has been attending for a number of weeks. It is a pleasurable way of teaching the child to use earphones, for until this has been achieved the child cannot be tested with our speech audiometer.*

Some children require to be conditioned at home. For these we have many animal toys and peg-toys which can be used to demonstrate to the mothers game-methods of conditioning their little deaf children to various sounds. All of these games are similar in their basic purpose and method which is that by means of a slight reward the child learns that a peculiar stimulus he receives (namely sound) has a meaning.

These four schemes arouse no antagonism or fear. The time consumed in testing is sometimes great but the reward is greater.

For the investigation of the hearing of the infant in arms we have a duplicate of the Johns Hopkins skin resistance machine. One of our Fellows is engaged in a three year investigation of the Baltimore claims.[3]

We have no use for watch tests, whisper tests, tuning fork tests or the monochord. In all our testing of the hearing of young children we do not use any test which cannot be performed in a very few minutes or repeated exactly many times on different occasions. Nor do we use any test which does not command the child's interest, or

*For those children who enjoy it thoroughly we have a much smaller portable loud-speaker arangement so that a parent can find out whether the small apparatus would be of use in her home.

which frightens him, or which does not arouse his co-operation—or which fatigues him. We never forget that the very young child is unable to concentrate his attention for any sustained period.

SPEECH TESTING

Older children we test for hearing of speech by a very simple method. We use a high-fidelity gramophone audiometer specially designed in Professor Solandt's department at the University of Toronto. We use the Western Electric recording K.S.6091 popularly known as the fading-numbers record. The patient replies not by writing but by speaking the numbers he thinks he hears.

By the use of this record the patient hears words with which he is thoroughly familiar. This record played on an ordinary gramophone cannot be used to test cases which have more than thirty decibels loss. As many of our patients are more deaf than this, amplification must be used. Our high-fidelity machine enables us to use this record to test patients with a loss of as much as eighty-four decibels.

Our machine has excellent attenuators—but we believe that the use of these in measuring hearing is inferior to the use of attenuation done in the recording.

We are convinced that a more useful estimation of hearing can be obtained by testing a young child with speech than by testing him with pure tones. We are satisfied that numbers are superior to common words and sentences. Nonsense sentences and meaningless speech sounds we have altogether discarded.

We do not use speech audiometry to check the validity and reliability of other hearing tests. Speech reception thresholds and discrimination scores have no value whatever in the testing of children's hearing.

DEFENSE OF THE FADING NUMBERS TEST

It is necessary to say a few words in defence of the fading-numbers test. In the first place it has the advantage of being a recorded testing device. Because, as I shall explain presently, I believe that live-voice must not be used in the measurement of hearing the only alternative is to employ testing material that is recorded.

In the second place an otologist is keenly interested in every test which brings him information he can use for his patient's better-

ment. The mass of the tests now used in audiology are of no practical value to him. His crying need at present is for a method which will provide data which can be compared with re-examination data. I believe that he can obtain this by using the Western Electric K.S. 6091 in a high-fidelity reproducing unit.*

Moreover it is the opinion of Hallowell Davis[4] that the use of the list of numbers in the Western Electric fading-numbers recording properly administered is a measure of the hearing loss for speech of the same precision and reliability as the pure-tone test of a clinical audiometer.

CONDEMNATION OF LOUD-SPEAKER FREE-FIELD TESTING

Except in special cases we advise against the use of loud-speaker free-field testing in any hospital clinic devoted to the hard-of-hearing and especially in such a clinic for children. This advice may cause amazement because this scheme is the predominant feature of nearly every such establishment set up in recent years. We believe that this technique is inaccurate for measurement of changes in hearing for a number of reasons.

The first important fault is that it makes use of live-voice.

Speech is influenced by the breath stream from the lower respiratory tract, modulated by the vocal cords, varied by the articulators (lips, jaw, tongue and soft palate) and its overtones are selected and suppressed by the vocal cavities (nasal, oral and pharyngeal). The whole process is governed by the brain. It is obvious that these basic speech processes cannot produce the same sounds for a second testing of the hearing for measured comparison unless they are in the same physical condition and are used in exactly the same way. Even if a very practised technician can with the help of a voice-level-meter learn how to control the intensity of his voice to an error as low as three decibels, that does not make it possible for him to make his tests constant in intensity for more than the same day. Nor does that prevent him, six months hence, from having got out of his bed on the wrong side, or from an intimate sorrow upsetting his laryngeal control, etc. etc. With such things to contend with, his testing procedure, six months hence, may not be the same as it is today.

The intelligibility of speech does not depend solely on the intensity of the speaker's voice—it depends also on its pitch and its

*The attenuation in the K.S. 6091 is in decibel notation.

timbre. The effort of these on the visible patterns of speech can be quickly discerned by a study of the illustrations in "Visible Speech."[5] There too can be seen the variation in the visualization of the same sentences and the same words. It will be apparent that the sound pattern of speech continually changes.

The next disturbing fact is that all words spoken naturally and in sequence do not have the same physical power. In addition even when carefully monitored to the same effort of speech, all words are not equally intelligible.

There are other difficulties in loud-speaker free-field testing. The acoustic properties of the sound-proof room in which the loud-speaker is used are amazingly difficult to keep constant for subsequent testing:—everything in the room must be exactly the same as was used at the previous test and in exactly the same position: the patient must wear the same clothes. If not—the Western Electric Company has proven that the tests are not comparable.

The obvious conclusion is that live-voice testing *for measurement* is a delusion.

When one contemplates the many thousands of dollars which have been expended in such units and in paying the salaries of the required personnel one wishes for the tongue and pen of a Jonathan Swift.

USE OF PURE-TONE AUDIOMETER

We do not attempt to test a child with the pure-tone audiometer until much later in his clinical life. We do not use it until the child's fears have been eliminated, until he trusts and obeys his instructor.

In using the pure-tone audiometer we have stopped using the push button. The action of pressing a button is distracting and takes time. We teach the child to raise his finger when he hears the tone and to drop it when the sound ceases. He sits with his back to the examiner and the latter watches the action of the finger and from it judges the patient's interest, attention, co-operation and alertness. To obtain a good audiogram on a young child is quite an art.

Some suppose that speech tests performed with selected word-lists are useful to show up losses of hearing in different parts of the frequency range and check the validity and reliability of the pure-tone tests. This we do not believe. The best way to test frequency

hearing is to use frequencies as stimuli. One cannot infer a patient's hearing of speech from a pure-tone audiogram.

We make all tests very short. The time cannot be stated exactly. A young child ought not to be in the sound-proof room longer than a few minutes at a time. Loss of interest on the part of the child will be shown in the gradual drooping of his signal finger, a delay in response and the swinging of his foot. As soon as the child's interest or co-operation flags we stop the test and leave the room.

Diplacusis measurements, tinnitus matching, recruitment evaluation, loudness balance, pure-tone averages and practically every other test to be found on audiological work-up charts are also useless in the testing of children's hearing.

Older, very intelligent children sometimes require modification of our regular testing techniques as also do young mental defectives. The amount of masking which is necessary varies. These modifications are not stereotyped but are made on the spot by the otologist. The latter is qualified to do so whereas an audiologist would not be.

IMPORTANCE OF LESIONS OF PERCEPTIVE APPARATUS

It is widely believed and taught that every lesion of the perceptive part of the hearing apparatus is irreversible and therefore incurable. It is maintained that histo-pathology proves beyond doubt the truth of this statement. Because of this the effort is made to diagnose lesions of the perceptive apparatus. Having made this diagnosis any attempt to ameliorate the condition by treatment is considered futile and even dishonest.

There are so few hearing tests which can be interpreted with reasonable certainty in terms of causative lesions that the presence or absence of tone and inflection in a young child's voice is of great diagnostic importance. A few excellent papers notably those by Kobrak[6] and Guild[7] have proved the inadequacy of many of our tests.

Therefore we should stop concentrating on lesions of the internal ear and instead we should devote our attention to finding variations in the patient's hearing. Variation can occur with both conductive and perceptive lesions. If the hearing really varies the underlying lesion is not yet permanently fixed and should be amen-

able to treatment. At present there is no test which demonstrates variability as effectively as the speech test.

We ought to know better than to assume that every lesion of the perceptive apparatus is incurable. For there are lesions of other nerves which recover, such as those of the optic nerve; a large scotoma may diminish in size until it is almost gone. It is therefore safe to assume that lesions of the perceptive apparatus of the ear are, in their early stages at least, reversible and amenable to treatment.

Let me give you an example. A six-month old patient of ours was a candidate for adoption. There was no evidence of any hearing and no ocular response to douching with cold water. We advised against adoption and as our findings remained unchanged we continued to do so. However at eighteen months his attempts at babbling were not flat and toneless and so we said there was still hope. At the age of two years and one month the child commenced to put three and four words together.

We are in the infancy of an understanding of the problems of perceptive deafness, but I cannot state too positively that we should be very slow in saying that such a child, who apparently has a serious perceptive lesion, cannot recover.

The number of patients whose hearing is defective because of a congenital lesion is small. The vast majority are hard-of-hearing because of disease usually infection. It is of fundamental importance that the otologist recognize the earliest signs of the disease and arrest the process at that stage if he can. He must watch the progress of the patient by measuring the hearing of the young child—a procedure which this paper has shown to be very difficult.

He should approach the problem with the knowledge that the child possesses immense powers of recovery.

Earlier in this paper the great importance of the history and of the examination were touched on briefly. The treatment depends on the facts so established. Whether a mother has noticed signs of earache or nasopharyngitis or not, an abnormal eardrum shows the otologist that both of these conditions have been present. The nasopharynx can only be seen by using the nasopharyngoscope. The examination must be done after adequate shrinking and desensitization and in the erect position. Examination in the supine position

under general anaesthetic is futile because bubbles of moisture absolutely prevent accurate observation. It is amazing how efforts to use the nasopharyngoscope on a small child will deflate a man's ego. Realization of the difficulties involved is the great reason why some clinicians avoid using it.

A serous effusion in the middle ear is very common—a blocked Eustachian tube probably much commoner. But a young child cannot describe the accompanying sensations. When the middle ear is filled with serum the drum may show little more than a faint pinkness—there may be adequate lustre. In some cases the fluid only becomes apparent after the use of the Eustachian catheter and inflation is the only way of curing the condition. To be able to use the catheter in a young child and to be able to continue to use it is an art. It requires a gentle touch and patience on the part of the otologist and trust by the little patient which is not won easily. In many clinics the teaching is to avoid catheterization. Some of our young clinicians, so taught, proved, by measurement, the efficacy of this treatment and they now know that what they were taught was incorrect. There is no need to fear that inflation of children's eustachian tubes will be abused. The danger, on the contrary, is that most otologists will abuse inflation by neglecting it.

The treatment of recurring nasopharyngitis depends first on the clinician's accurate diagnosis of the cause. Some of the possible causes are the adenoids, unfortunate hypertrophy of other lymphoid elements, or even sinus trouble. The treatment of each of these to a successful termination requires qualities in the otologist of no mean order. They are qualities which the surgically-minded specialist is most unlikely to possess.

Failures in treatment are many—but there are also spectacular successes. Even partial successes are viewed with gratitude by the parents.

Visit what clinic you will and you will be impressed by two things. The time devoted to audiologic technique and work up is very great, but that devoted to history taking, examination and treatment of the child is brief.

This is not as it shoud be. The most important part of all, and the part that should be great is the estimation of the child's hearing ability by the otologist and his continuing attention to the problem.

The difficulties of all phases of this work in young children cause many otologists to shirk it and pass it off to the audiologist, to the nurse and to the young doctor. But this is quite unworthy. The younger the child the greater the difficulties. The latter present a challenge which should be met.

It should be our ambition in this great society to so arouse the medical profession to the beginnings of deafness that early attacks upon the hearing apparatus of the child will be arrested and the changes that have been produced wiped out while they are still reversible.

The way to correct the first fault noted in this paper—namely —evasion—is simply to bring back the otologist to his rightful place and set him to work at diagnosis. He requires to discard research techniques which are out of place in a clinic. He must use methods of testing suitable for children.

To correct the second fault—neglect—the otologist should apply the methods he uses for the adult to the child in spite of the much greater difficulties. Let him persevere in his treatment. Let him not overlook his great ally—the vast recovery powers of the child.

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DISCUSSION

DR. DOUGLAS MACFARLAN: I had the pleasure of reading this paper before it was given and I am so sorry Dr. Wishart had not time to read it in full because I feel that it is most important that we take our places as otologists in this picture. You cannot leave the otologists out of the picture of the congenitally deafened child and the hard of hearing child.

The very first thing in either of these types of children is to get an otological diagnosis. As to the diagnosis of congenital deafness, we cannot call them all that because we don't know how many are congenitally deaf and how many have acquired deafness before language. These children are not always brought to us before the age of two. They come to us at a time when parents find they are not learning speech when they should be. As Dr. Wishart and Dr. Hill wisely pointed out, we must do something early and not leave them until five or six years of age at which time they are acceptable to a school for the deaf.

The technique of handling these children is a matter in which you have to train yourself. If you have any genius or ingenuity at all you will very quickly see the problem. Th first two problems to overcome are the restlessness and the inattention. When you get past these two obstacles, you begin to train them in conditioned response.

In the old days we were accustomed to use a buzzer loud enough to rattle an earphone in the ear, and with that buzzer on them they could be taught to raise their hands, because they felt the vibration as when they heard it. When they responded correctly by raising the hand you could reduce the intensity of the sound, dropping it below the tactile threshold. If they still reported correctly, they were hearing, not feeling. An audiogram could then be taken. Now, there are new methods, Ewing-Hallpike-Wishart (the so-called "Peep-Show"), and the latest method "the Jack-in-the-Box." The latter is described in the February 1952 Archives.

Both methods depend upon tying in a visual response with an auditory perseption, when we are interested only in the auditory factor. Yet, this seeing of something interesting and amusing will affect their trainability. So much the better.

You can in the office develop with most of these children an immediate opinion as to whether or not there is residual hearing, and after all, that is the important thing. We want to know if these children have residual usable hearing. Strange to say, 80% of them do, and you can put a hearing aid on them before you have an audiogram. You will be very much surprised how you will awaken the dead auditory attention which at times is very profound. One of the early responses which you can get in the office, is the pleasurable response when you put phonograph music on and you see the little faces light up. When you get the pleasurable response, you know you have something to work on. It is very strange how much language they can learn in a very short time, in a year's time.

Dr. Wishart did not have time to develop all the phases of this work, of how much we have to depend upon the education of the parents and the teamwork with educators. I cannot say we will get so much teamwork from the psychologists, because the child psychologists who know the deaf child, are few and far between. You will have to be your own psychologist in this work.

One thing that should be mentioned in the handling of all these cases is this, the thwarting caused by the deafness undoubtedly alters them very early and by four or five years of age they are incorrigible children and very often they are supposed to be mentally deficient. That behaviour problem will clear up, or nine-tenths of it will, under training, and they will be acceptable at least to a day school for the deaf if you put a hearing aid on them so they can be reached by residual hearing. It is absolutely wrong to condemn these children to a diagnosis of amentia or aphasia without first going through a preliminary training period in which you try to reach their hearing.

I think the biggest contribution of this paper of Dr. Wishart's is that it shows it can be done, and Dr. Hill's paper too shows it can be accomplished and without too elaborate equipment.

The most important equipment that we need lies above our ears. Most essential is a spirit to go ahead and investigate. The procedure is not too time consuming for us. Make the diagnosis early, put on a hearing aid, and get proper training program under way.

DR. KENNETH DAY: I think much of Dr. Wishart's criticism of audiology departments is deserved. However, I am actively associated with an audiology department at home and I am very proud of it. It has grown into a very busy department. It does research. It trains teachers. There are a number of classes for the hard-of-hearing pre-school children and their parents. There is a university program of lectures for the parents of hard-of-hearing children with a series of twelve lectures twice a year and attendance as high as one hundred. The parents have organized an association among themselves which is active in advertising the work which is being done in the city. I think it is quite wonderful.

One of the most important functions of an audiology department is the detection of residual hearing in the so-called congenitally deaf children and the institution of early training. If this can be done at one or two years of age it is far better than at three or four. These children must learn to listen with their eyes and their brains by auditory training and speech reading. The younger they start the quicker they learn.

One point I should like to emphasize is the importance of routine otologic examination of all infants. Many get secretory otitis media or have nonventilated middle ears due to improper feeding. I wonder how many children thought to be congenitally deaf may not have had their ears insulted during the first few months of life.

A good audiology department should be under the direction of and co-ordinated with a department of otology. Teamwork between these departments is essential. Cases should not be accepted for study which have not had an otologic examination and diagnosis first. Moreover, an audiology department should not be allowed to become a dumping ground and required to do hearing tests and fit hearing aids by doctors who are too lazy or disinterested to do this work themselves.

DR. STACY R. GUILD: Dr. Wishart mentioned in the early part of his verbal presentation not only the peep show but the psycho-galvanic skin reflex. I hope the complete paper contains more about the latter procedure, because by it, as Dr. Hardy and Dr. Bordley of our Department have stated, valuable information can be obtained at a very early age.

Anatomically speaking why is it so necessary, if you are going to have good speech, to get the educational program going early? The normal hearing child

learns to speak during its first three years. During the early years, myelinization of the pathways concerned is taking place. It is exceedingly difficult later to get the same amount of voice for the same, or even a much greater, amount of effort.

Finally, a few practical points for those of you whom I hope are going home and emulate what Dr. Hill does, either simply or on a more elaborate scale. Some people can get along with children and some cannot. I happen to be one of those who can get the cooperation of children. From my experiences I recommend that you do not wear a white coat when meeting a child for the first time. I go in wearing my street coat or with my coat off, in my shirt sleeves if it is warm weather. A second suggestion is to carry a roll of small candies in your pocket. Another aid in gaining cooperation is to have a battery of tuning forks on a little low table and to let the child wander over and start banging them around a bit while you are writing some notes. He won't do it but once if he does not hear something. If he wishes to, let him play with them. First, though, warn the mother not to stop him. If he hears a fork after he has banged it his face lights up and you not only learn something about his hearing but also make a friend by helping play his game. Then he is willing to play your games.

DR. MOSES H. LURIE: One of the remarks Dr. Wishart made earlier in his paper referred to one of mine on the treatment of deafness in children that I made before the Society some years ago. I agree with him entirely that the deafness should be detected early so that the child can be trained early because a few of us realize that the child with any hearing develops a symbolic relationship of the sounds heard with the outside world, and thus lays the pattern of his thoughts for the rest of his life.

This symbolic relationship has been recognized by a number of philosophers and one of the best books written on the significance of sound in human civilization has been written by a disciple of A. N. Whithead. Suzanna Langer wrote a book called "Philosophy in a New Key," in which she points out that our civilization is based on the symbolic significance of sound and speech. In it is also presented a very beautiful description of how a child learns to speak. This book has created a great deal of interest among psychologists and philosophers, and has been published by the Mentor Library, so for thirty-five cents you can get one of the modern conceptions of what hearing means to modern man. In there also I think you will find a good many things that Dr. Guild has talked about, and what Dr. Wishart has discussed. He has arrived at similar conclusions with his keen deductions from trying to find out how children hear and how speech develops in the young child.

DR. D. E. STAUNTON WISHART: Answering Dr. Guild, we are only learning how to use the psycho-galvanic skin resistance machine. It will be three years before we have results to report.

I want to emphasize that what I read to you had to do with our painstaking investigation of the hearing of very small hard-of-hearing children. The part that I did not read is what we otologists must do for the child who has only remnants of hearing. To do this work there should be facilities for testing hearing in the clinic for the clinicians to use. They have no measurement of the result of their efforts unless they have a method of testing the hearing which is simple and quick and which they do themselves.

LXVIII

A TINNITUS IDENTIFICATION TEST

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Tinnitus is an otologic symptom. It has many characteristics and can occur as the result of many causes. It is not a disease or syndrome. It is a subjective phenomenon common to many diseases and should be viewed in the light of subjective symptomatology in differential otologic diagnosis.

DEFINITION AND CLASSIFICATION

Webster defines tinnitus as a ringing, whistling, or other sensation of noise which is purely subjective. The word is derived from the Latin, "tinnire," meaning "to jingle."

Basically two types of tinnitus must be distinguished; tinnitus aurium and tinnitus cranii (tinnitus cerebri).

Tinnitus cranii is frequently confused with tinnitus aurium and may actually co-exist with it. Tinnitus cranii is a non-localized subjective sensation of sound which is usually diffusely in the head and has a non-specific quality. It is frequently described as a roaring or rushing sound not directed to the ear region. Its diffuse character may be confused with somatic sensations of the neck and upper thorax and, indeed, may be due to vascular phenomena in these areas as well as intracranially. Most usually, tinnitus cranii is due to organic or functional intracranial vascular disease and is a medical neurological problem, one which does not participate significantly in otologic diagnosis.

Tinnitus aurium, on the other hand, is usually localized to one or both ears and is subjectively described with some specificity by the patient.

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Tinnitus aurium must be subdivided into two sub-types each of which has been given a number of names. These two principal sub-types and their synonyms may be described as follows:

1. Subjective tinnitus (static, non-vibratory, true, intrinsic). Subjective tinnitus is the subjective cortical perception of auditory sensations inaudible to anyone but the patient. These sensations are most usually due to auditory paresthesias from any location within the auditory pathway.

2. Objective tinnitus (dynamic, vibratory, false, extrinsic). Objective tinnitus may be defined as the subjective cortical perception of auditory sensations, potentially audible to an examiner as well as to the patient.

Inasmuch as objective tinnitus is a relatively rare phenomenon, its characteristics and significance in otologic diagnosis will be briefly surveyed in order to limit the remainder of the discussion to the principal type of tinnitus encountered in otologic practice; namely, subjective tinnitus.

Objective tinnitus, which has been very aptly described by Dr. Fowler, Sr., as vibratory tinnitus, is either vascular or muscular in origin. The vascular types of objective tinnitus are usually due to some type of arterio-venous communication producing an audible bruit or murmur which can be heard with or without amplification. The muscular types of objective tinnitus are due to contractions of tympanic or tubal muscles, usually in bizarre or atypical rhythm. The mechanism for such contractions is exceedingly complex in etiology. Muscle spasms, functional tics, and other neuromuscular phenomena related to metabolic and psychosomatic states play a part in this relatively rare group of cases.

THE ETIOLOGY OF SUBJECTIVE TINNITUS

The etiology of subjective tinnitus can be discussed from the standpoints of anatomic location and pathologic change. Subjective tinnitus may arise from any location within auditory pathway from the external ear to the auditory cortex. The pathologic state responsible for the genesis of the tinnitus may be any histopathologic abnormality in the auditory pathway. Consequently, the potential causes for tinnitus are innumerable and defy simplification. Tinnitus is a common symptom of many otologic diseases.

The following table suggests the multiplicity of anatomic and pathologic factors important in tinnitus etiology.

ETIOLOGY OF TINNITUS

(Differential Diagnosis)

ANATAMIC LOCATIONS	PATHOLOGIC LESIONS
External auditory canal	Anemia
Tympanum	Hyperemia
a. Memb. tymp.	Edema
b. Tymp. muscles	Serous exudate
c. Ossicles	Mucous exudate
d. Tymp. plexus	Purulent exudate
e. Tymp. vessels	Hemorrhage
f. Eust. tube	Inflammation
g. Fenestrae Rotund and Ovale	Allergy
Cochlea	Neuronitis
a. Perilymph	Necrosis
b. Endolymph	Fibrosis
c. Organ of Corti	Tumor
VIIIth Nerve	Aneurysm
a. Spiral ganglia	Capillary fragility
b. Trunk	Stasis
Intracerebral	Sludging
a. Ventral and dorsal nuclei	Vascular spasm
b. 2nd order neurons	Vascular sclerosis
c. Med. Gen. body	Vasomotor paralysis
d. 3rd order neurons	
e. Cortex	

THE SIGNIFICANCE OF TINNITUS IN THE DIFFERENTIAL DIAGNOSIS OF OTOLOGIC DISEASE

Otologic diseases are characterized by a few specific findings and symptoms, the most common of which are otorrhea, otalgia, vertigo, deafness, and tinnitus. The complexity of the anatomic

pathway involved in auditory physiology makes the assessment of every symptom important in otologic diagnosis. Consequently, any refinement in description of each of these few cardinal symptoms, will be of importance in the differential diagnosis of otologic disease. We have attempted to refine our diagnostic techniques to a greater or less degree. Our studies of the bacteriologic and cytologic aspects of otorrhea, our quantitative and qualitative studies of hearing and vestibular functions, bear witness to these attempts. Tinnitus is an extremely common, and indeed profound symptom, in many otologic diseases. Yet we have been unable to obtain comparative qualitative and quantitative data on it in the past.

In that vast unknown called "nerve deafness," which comprises many diseases occurring in the intricate neural pathway from the endolymphatic labyrinth to and including the auditory cortex, we have few localizing opportunities in differential diagnosis. The threshold audiogram, recruitment tests, speech audiometry, and correlated vestibular studies have given us some insight into "nerve deafness." Tinnitus is an important symptom in lesions of the neural auditory pathway. The identification, accumulation, and classification of discrete characteristics of tinnitus in these cases, when correlated with other otologic data and later with pathologic findings, may shed much light on the differential diagnosis of "nerve deafness." Recent studies have shown that much nerve deafness originates not only in the organ of Corti, and in the cochlear ganglion, but in the nuclei, ascending pathways and possibly in the thalamus and cortex. Present audiometric technics do not suffice in the segregation of anatomic locations in patients with nerve deafness. It is conceivable that correlation of audiometric data with tinnitus analysis data may yield such localizing information.

TINNITUS AS A SUBJECTIVE PHENOMENON

When one enters that somewhat esoteric domain known as subjective phenomenology, one is immediately struck by semantic difficulties and variability in descriptions of subjective sensations.

When a patient is asked to describe a subjective sensation such as tinnitus, the accuracy of the description will depend upon many factors; including native intelligence, analytical subjective ability and degree of semantic articulateness. Of specific importance in the description of tinnitus, is the acquaintance of the patient with acoustic or musical terms. A patient with slight musical experience will have

difficulty in pitch discrimination and in pure vs complex tone differentiation. His conception of intervals, octave and other scale relationships will also usually be poor. Consequently, the validity of a subjective description of tinnitus depends largely upon certain special qualifications on the part of the patient. Inasmuch as most people are relatively inexperienced in acoustic or musical terminology, it is difficult to obtain clear descriptions of the components of subjective tinnitus.

METHODS OF STUDYING TINNITUS

When patients with tinnitus are asked for a description, a loose indefinite answer is usually obtained. Examples are:: "I hear steam escaping; I hear bells ringing; I hear whistles blowing; I hear a roaring or buzzing in my head; I hear a sound like a seashell held up to my ear; I hear sounds of a squeaking door; I hear the sound of a babbling brook; I hear the noise of a radiator; I hear the noise of an ocean roar; I hear radio static; I hear clicks or rumbles of a dynamo, or a generator."

Only a few patients will be able to accurately compare the tinnitus with a pure tone on the audiometer. In acoustic trauma cases this is fairly frequent because of the apparent anatomic specificity of cochlear damage produced by noise.

It is thus, only the scientific or musically articulate patient who will describe his sensations with some degree of accuracy. Such a patient in the acoustic trauma case will be able to localize his pitch and intensity characteristics with considerable skill. In the more complex types of tinnitus encountered in otosclerosis, labyrinthine hydrops, and acoustic neuromata, such a patient will frequently volunteer an attempt at a more complex description in such acoustic terms as preponderance of primary tones with accompanying tones, octaves and over-tones. Unfortunately, too few descriptions of tinnitus have been obtained from such patients.

A number of patients with varying otologic diagnoses, who had either sufficient musical or acoustic experience were able to give data useful for the construction of a tinnitus identification recording.

This original recording, and its subsequent modification, was artificially created by sound technicians to mimic as closely as possible the acoustic components described by patients with tinnitus.

The acoustic components were derived from the use of combined oscillators and from material from a sound effects library.

The following recording will illustrate some of these "tinnitus reproductions." This preliminary attempt is a rather elementary and probably crude group of reproductions. Nevertheless, in starting such a project, some beginning was necessary to obtain basic comparisons for subsequent alteration.

TINNITUS IDENTIFICATION RECORD

1. 2000/5000 Cycles, 2000/8000 Cycles.
2. 2000 Cycles, 2150 Cycles, ON-OFF.
6. 2000 Cycles with beats, slow fades.
8. 3 Oscillators, 2000 Cycles, 2150 Cycles, 2290 Cycles.
11. "White noise."
12. Static.
13. Cross-modulation, 5600 and 6000 Cycles, not overloaded.
14. Cross-modulation, 5600 and 6000 Cycles, overloaded.
15. Inter-modulation, 60 and 1000 Cycles, not overloaded.
16. Inter-modulation, 60 and 1000 Cycles, overloaded.
17. Escaping steam noise.
18. Escaping steam noise.
19. Heavy ocean noise.
20. Heavy surf noise.
22. Water faucet hum.
23. Radio static and whistles.
24. Stethoscopic heart beats. (From Col. record #11007-M).
27. 200 Cycles and 2000 Cycles, overloaded with 200 Cycles pulsating 70/min.

METHODICAL ANALYSIS OF TINNITUS

A methodical analysis of tinnitus should be an integral part of the otologic examination, particularly in clinical research. The following method of analysis is suggested as a somewhat orderly technic in an attempt to obtain useful data.

a. *Subjective statement of patient.*

The patient is requested to describe his tinnitus in his own words to the best of his ability without any specific questions.

b. *Subjective analysis.*

The patient is then questioned for the following specific data:

1. The general location of the tinnitus. Is the tinnitus *within* the patient or does it seem to be somewhere in the room? Occasionally the patients will insist that the tinnitus is not in the head, not in the ear, but to the right or to the left of the head and apparently projected several inches or feet away.

2. The somatic location of the tinnitus. Is it in one or both ears and if so, superficially or deeply; or is it in the head, and if so, in what part of the head?

3. If the somatic location is somewhat diffuse and variable, the patient is asked to relate the specificity in regard to loudness. If the tinnitus is stated to be in the head and ear, the predominant intensity is elicited.

4. *Time relations of tinnitus.* Is the tinnitus louder during certain times of the day? Is it louder at work or at home? Is it louder at certain times of the week? In women, does it vary with menses?

5. *Positional relations of tinnitus.* Does the tinnitus vary with positions of the head or body? Does it increase on stooping? Is it relieved by any specific position of the body or head?

6. *Characteristics of the tinnitus—acoustic analysis.*

a. If the tinnitus is a pure tone, it may be identified on the audiometer. For these purposes, the ordinary fixed frequency audiometer is inadequate. The sweep frequency audiometer is necessary to accomplish such specific tone localization. By this same technic the intensity and the masking intensity required to mask the tinnitus can be determined.

b. If the tone is complex, an attempt is made at audiometric analysis, in terms of dominant and accessory tones. Admittedly in most patients this is a difficult if not impossible task but in musically articulate patients it can frequently be accomplished.

c. *Tinnitus identification test—recording.* In all cases where tinnitus is complex in nature, the patient is requested to listen to the tinnitus identification recording in an attempt to locate an acoustic component which might compare with his subjective phenomenoma. It is explained that although the pitch in the recording may be different from that of his tinnitus, we are interested in knowing whether the tone quality, combinations, or effect in any way resemble his subjective sensations. Many patients will respond to the tinnitus identification record by picking out two or three components and giving dominance to certain of these components.

All psychological tests carry the burden of interpretive, emotional and other variables. This tinnitus comparison or identification test is a subjective test and falls heir to these inadequacies, but so does the threshold audiogram, and the test for recruitment.

It is surprising to note the frequency of positive comparisons in the short period of clinical trials. This can be illustrated by the following cases.

CASE HISTORIES

CASE 1.—Dr. ARB, age 56, otolaryngologist. Progressive bilateral perceptive deafness. Threshold loss 50 db left and 45 db right. Moderate recruitment bilaterally. Hears tinnitus bilaterally similar to No. 18.

CASE 2.—Mr. W. S., age 61. Chief complaint—tinnitus, left. Bilateral perceptive depression 35 db. Tinnitus similar to No. 12; occasionally similar to No. 19.

CASE 3.—Mrs. A. G., age 56. Chief complaint is bilateral tinnitus. Examination revealed bilateral perceptive sloping curve of presbycusis type with predominant high frequency impairment. Her tinnitus was quite similar to No. 18.

CASE 4.—Mrs. R. G., age 42. No complaints until twenty-four hours after administration of 40 grains salicylates. Onset of severe bilateral tinnitus and hearing impairment. Tinnitus and deafness disappeared when drug stopped but returned when drug resumed five days later. Drug stopped again. Otologic examination following second episode revealed only slight high tone impairment audiometrically. Tinnitus was matched by No. 11 and No. 12 on the tinnitus identification record.

CASE 5.—A. H., age 56. Chronic bilateral otosclerosis, advanced, with neural degeneration. Tinnitus analysis revealed intermittent bilateral "ringing" type. On tinnitus identification record, close similarity with No. 6, No. 12, and No. 24 was noted.

RESULTS OF TINNITUS STUDIES

Insufficient data to attempt a correlation between the above method of tinnitus analysis and other otologic findings, have been obtained for definitive conclusions. Many clinical studies will be required for even presumptive conclusions. The final confirmation will await correlative clinical and pathologic studies of both temporal bones and brains of patients.

SUMMARY

1. Subjective tinnitus may be a symptom of disease anywhere in the auditory pathway.

2. Its elucidation and analysis are important in otologic differential diagnosis.

3. Subjective descriptions of tinnitus are usually indefinite and variable.

4. An elementary recording of "tinnitus mimicry" has been prepared for clinical investigation.

5. A methodical analysis of tinnitus has been proposed for use in present otologic research studies. Such a method may have clinical usage value in the near future.

6. A few illustrative cases are presented.

CONCLUSIONS

1. Tinnitus is an important symptom in otologic differential diagnosis.

2. Analysis of tinnitus is difficult without some comparative identification test.

3. A tinnitus identification test in preliminary pilot form is presented for use in tinnitus analysis.

DISCUSSION

DR. THEODORE E. WALSH: Dr. Goodhill's recording is the most intriguing sound we have heard. We have been trying for some time to match tinnitus with laboratory sounds in patients who complained of it. We have been able in many instances to match it fairly closely. The patient recognized the sound we put through the earphone as being something like the sound he is complaining of.

The thing that has interested me a great deal and I wonder if Dr. Goodhill has some information on it, is the subjective intensity of tinnitus to the patient. We have found people who complained bitterly of terrific noise in the ear and on measurement have found that the loudness of their tinnitus is only one, two or three db above threshold. The intensity is peculiarly small in actual measurement in decibels, yet some patients complain bitterly of it. I think the highest intensity we ever have gotten—and I think Dr. Lempert will remember this very well—was eight or ten db above threshold. This patient has been repeatedly tested and it comes out the same every time. The test is completely repeatable. I wonder if Dr. Goodhill could elaborate on the aspect of the problem of tinnitus.

DR. EDMUND P. FOWLER, SR.: I am afraid you rang the bell. I have a tinnitus right now in my left ear. I have had it on and off for many, many years. I formerly thought I was getting otosclerosis or some other condition but have never done much about it. I fades away at times and ceases for hours at a time. Last night coming up on that jittery train, without any sleep, probably brought it on today, that and the wonderful reception you gave me this morning.

But for once I agree with both the speakers. The sounds that were produced did not sound like anything I recognize. To me they did not seem even close to right.

The essayist said he obtained no beat with tinnitus. Many times I have had a tone of about 256 in my ear. I have nailed that down. I have rushed into the sound-proof room to see if I could not get beats with the tinnitus. Every time when I reach the sound-proof room the tinnitus stops. Now you see that must be some neurovascular thing. It must be some emotional thing, or something in the central nervous system.

What Dr. Walsh said about the loudness of the tinnitus I have said many times. It is true that you do have a valuable tool to work with if you can show that the patient by his own say-so says that his tinnitus sounds as loud as a certain sound which you are giving him which sound does not have to be exactly the same as his tinnitus. Usually you find it is only 3, 5 or 10 db in intensity. I have seen it up to 20. You prove to the patient that this is all they are hearing. You say to them, "Listen to the outdoor, street noises. There are louder sounds than those in your ear. With that you bring them up short and you start on your rehabilitation of the patient. I am not romancing about these reactions. I have had even old people with arteriosclerosis, heart disease get rid of their tinnitus in spite of arteriosclerosis. When they settled down emotionally the tinnitus went away. So I am convinced there is a large emotional element in it.

DR. KENNETH DAY: I think it might be in order for me to make a few comments since I have had three different types of tinnitus myself. I have either No. 18 or 19, the steam escaping. This is constant and has been present for as long as I can remember. It is not bothersome and I have to listen for it in order to notice it. Secondly, I frequently have a pulsating vascular tinnitus which

can be very annoying. I can usually suppress it by twisting my head into certain positions. Thirdly, I have had several attacks of a low-pitched roaring tinnitus lasting for several hours at a time. The pitch is around 100 cycles. I have tried to measure its loudness with the audiometer and it seems to be between 10 and 20 decibels above threshold. It is really terrific. I have walked the floor with it at night. I suspect it to be a vasomotor reaction in my right labyrinth, perhaps a tendency to an endolymphatic hydrops.

DR. VICTOR GOODHILL: May I repeat that this is a crude beginning at an attempt to mimic a subjective sensation. The selection of a 2000 cycle tone as a reference tone was purely arbitrary. This pitch standard may be varied in future editions of the recording. We may go to 4000 or 5000 cycles. The other components; namely, complexity, beats, and pulsations, may be more significant than pitch.

It is difficult to explain the discrepancy between the patient's distress and the slight intensity factors in some tinnitus measurements where the patient may complain about an intensity of 5 decibels. Perhaps the emotional level and related psychosomatic factors may color the tinnitus picture.

We are inclined to minimize the decibel as an expression of loudness and frequently must remind ourselves of its logarithmic nature. Thus, a 20 decibel increment in loudness of tinnitus can mean 100 times the pressure in dynes per square centimeter. Such a tinnitus level can be very distressing.

LXIX

THE OTOTOXICITY OF STREPTOMYCIN

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and

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The nature of the ototoxic action of streptomycin has been a matter for lively discussion and active investigation since the phenomenon was first described by Hinshaw and Feldman (1945). Using the crude preparations of the antibiotic then available for the treatment of tuberculosis, these authors observed in their original series of 34 patients one case of transient deafness and three cases of disturbed vestibular function, which they tentatively ascribed to a selective neurotoxic action on the eighth cranial nerve. Molitor and his collaborators (1946), who first noticed similar changes in dogs, suggested that they might represent either a labyrinthine or a cerebellar disturbance.

These observations immediately aroused the interest of otologists and other students of the nervous system, more especially because the toxic action of this important antibiotic seemed to be more highly specific than that of any of the few other drugs known to affect the vestibular and auditory systems. For this reason streptomycon might also be expected to furnish a useful experimental tool for the investigation of labyrinthine and cochlear function. Furthermore, with the relatively large doses which were originally used in tuberculosis therapy, the vestibular disturbance appeared at first to be a matter of serious concern and possibly a deterrent from the prolonged use of the antibiotic (see Minutes of the Third Streptomycin Conference, Veterans Administration, 1947). It gradually became

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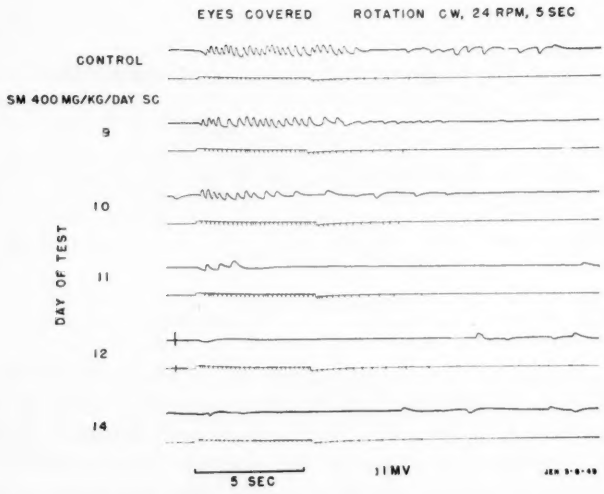

Fig. 1.—Electrical records of horizontal vestibular nystagmus in cat 1135, before and during treatment with streptomycin. The upper line of each pair represents the eye-movements, as recorded by means of the corneo-retinal potential, during and after rotation in the clockwise direction. The head was covered with a cardboard shield. Thus visual stimuli were excluded and no optokinetic response occurred. The lower line of each pair shows the 5 sec. period of rotation of the turntable, followed by deceleration to a stop. Strokes are for each 30 degrees of rotation. Ataxia was + on the 9th day of treatment, ++ on the 11th, +++ on the 13th and ++++ on the 16th.

clear, however, that a dose of 1 gm of streptomycin daily or even twice weekly gives therapeutic results not greatly inferior to those obtained with doses of two or three gms daily. At the same time preparations of increasingly high purity became available, and these factors, together with the introduction of dihydrostreptomycin with its lower vestibular toxicity, brought about a striking reduction in the incidence of vertigo and ataxia in the course of tuberculosis therapy. Dihydrostreptomycin however has been reported by a number of authors (Romansky, Katz and Glorig, 1949; Allison and Volk, 1949; and others) to cause hearing loss when given in doses larger than 1 gm daily or for longer than 120 days, and especially when administered by the intrathecal route in the treatment of tuberculous

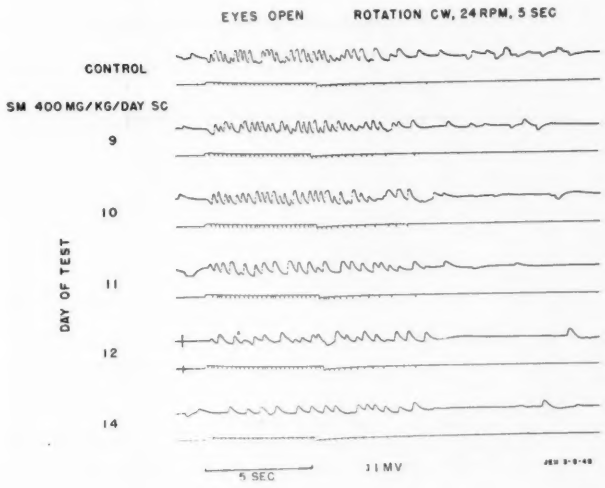

Fig. 2.—Electrical records of horizontal vestibular and optokinetic nystagmus in cat 1135. The eyes were not covered as in Fig. 1. Note persistence of optokinetic component when all vestibular response had been lost.

meningitis. Attention has been called to the fact that the toxicity of dihydrostreptomycin for the auditory system of the cat varies depending upon the purity of the preparation used (Hawkins, 1950) but the clinical significance of impurities in dihydrostreptomycin cannot yet be regarded as established. Finally certain new antibiotics, such as hydroxystreptomycin and neomycin (Hawkins 1951, 1952) have been found to be more strongly ototoxic for the cochlea than streptomycin or dihydrostreptomycin. It is clear, therefore, that ototoxicity can be an important factor in determining whether or not other new antibiotics will be suitable for clinical trial.

Thus, for practical as well as theoretical reasons, the study of the ototoxic action of streptomycin has been pursued in many laboratories and hospitals, both American and European. The question of the site of action has received particular attention. Among the authors who have held that the primary effect is central are Stevenson, Alvord, and Correll (1946); Winston, et al. (1948, 1949); Dix, Hallpike, and Harrison (1949); DeKleyn and Van Deinse

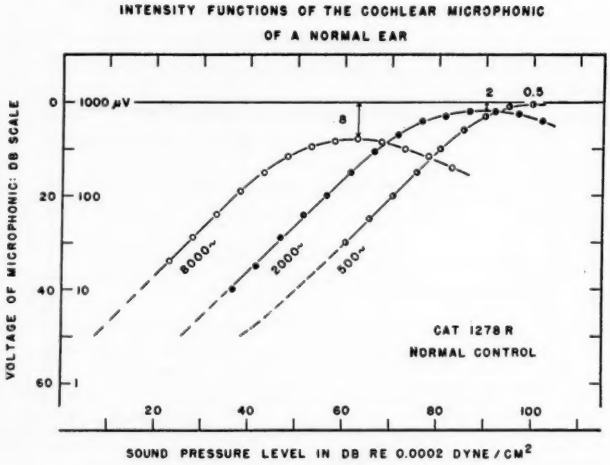

Fig. 3.—Intensity functions of the cochlear microphonic response of a normal ear. Figures above the curves show maximum response in decibels below 1 mv. Sound pressure levels as read by means of General Radio sound level meter with microphone placed in sound field directly over the cat's head.

(1950); and Christensen, et al. (1950, 1951). Floberg, Hamberger and Hyden (1949) claimed that the cells of Scarpi's ganglion showed toxic changes in streptomycin-treated guinea pigs. Glorig and Fowler (1947) and Vanderhaeghe (1949) on the other hand inferred from the persistence of nystagmus in response to galvanic stimulation when the response to caloric stimulation or to rotation had been abolished by streptomycin that the lesion must be in the end-organs. Independent histological support for this point of view was brought forward in 1949 by Caussé et al. and by Berg. Moreover, Rüedi et al., who in 1948 described central lesions in streptomycin-treated guinea pigs have recently (1951) confirmed in this species the end-organ lesions seen by Caussé in the mouse and by Berg in the cat.

Because of minor differences in behavior between streptomycin-intoxicated rats and those bilaterally labyrinthectomized, I was at first inclined to believe that damage to the peripheral end-organs alone could not account for the vestibular effects, but that the func-

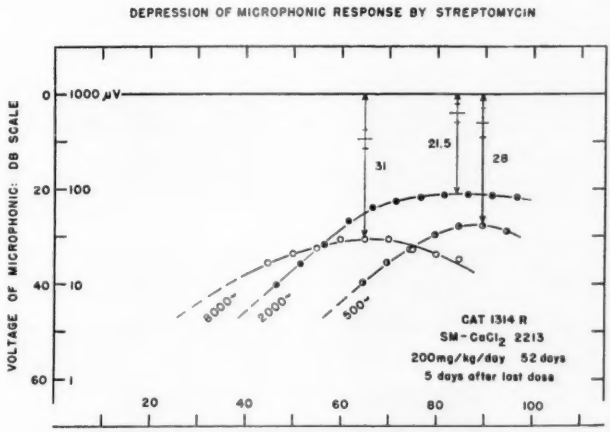

Fig. 4.—Intensity functions of the cochlear microphonic response in cat 1314, showing depression by streptomycin. Horizontal bars above curves show average maximum responses for 21 normal control ears, with standard deviation.

tion of the central pathways must also be affected (Hawkins and O'Shanny, 1949). Later, however, I was able to confirm in the cat the persistence of galvanic nystagmus in severe streptomycin intoxication. Further proof that the primary lesion must be peripheral was obtained in the study of the electrical phenomena recorded from the round window of the cochlea, which showed that the cochlear microphonic response is depressed when hearing is affected by streptomycin. An account of these experiments was published in 1950. In the present paper we shall report on functional and anatomical studies in eight streptomycin-treated cats, including two of those discussed in the 1950 publication, and in two normal control animals.

Methods: Carefully selected, healthy adult cats were treated with daily subcutaneous injections of streptomycin calcium chloride complex in amounts equivalent to 100, 200 or 400 mg of streptomycin base per kg of body weight, for periods up to 60 days. The potency of the preparations used was approximately 730 micrograms

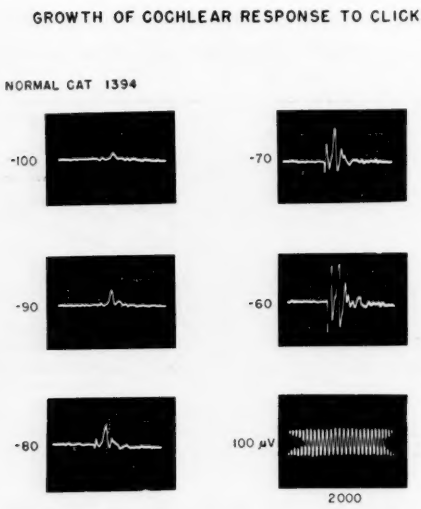

Fig. 5.—Responses to clicks recorded at the round window in a normal control cat. Figures show intensity of clicks in decibels below arbitrary standard level. Calibrating voltage shows 2000 cps, 100 μv rms.

of streptomycin base per milligram of substance, corresponding to a purity of about 95%.

Vestibular function was treated at frequent intervals before, during and after the period of treatment by recording the horizontal nystagmus in response to rotation on a motor driven turntable. For this purpose small metal electrodes were pasted to the shaved skin on either side of the eyes to pick up the apparent variations in corneo-retinal potential as the eyes moved from side to side in nystagmus (Schott, 1922; Meyers, 1929). A brief account of this method was given at the 1947 meeting of the Otological Society.

Tests of cochlear function were made by the method of Wever and Bray (1930). By means of electrodes, placed one on the round window membrane of the cochlea and one on the margin of the skin wound, the cochlear potentials were recorded in cats anesthetized with pentobarbital. The responses to tones and to impulsive (click)

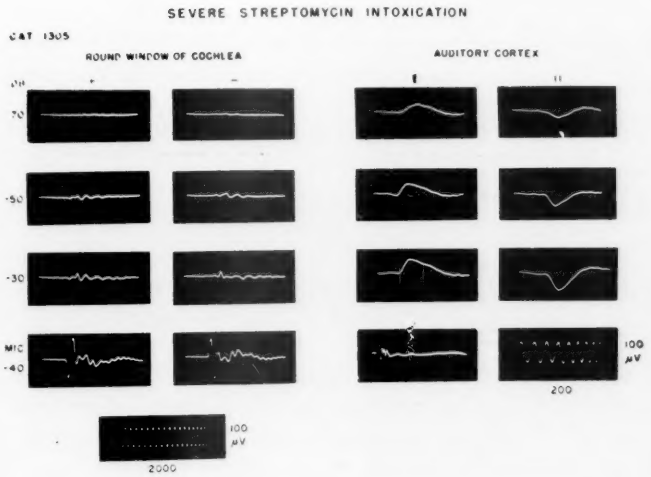

Fig. 6.—Responses from the round window and the ectosylvian cortex of streptomycin-treated cat 1304. Between the first and second columns the polarity of the stimulus was reversed to demonstrate small microphonic component. Auditory cortical responses: pattern I from anterior portion of middle ectosylvian gyrus; pattern II from mid-portion of the same gyrus (Hawkins, 1941).

stimuli were recorded by the means of the cathode-ray oscilloscope and their amplitude measured by a calibrating circuit.

At the conclusion of the cochlear tests, the carotids were cannulated and the head perfused from a reservoir bottle at a pressure of approximately 120 mm Hg according to the method of Koenig, Groat and Windle (1945), first with a solution of 5.6% gum acacia in 0.9% NaCl solution to wash out the blood, and then with a solution of 0.9% NaCl and 5.6% gum acacia in 10% formalin to fix the tissues. The temporal bones were removed, and after further fixation in Heidenhain-Susa solution, they were ready for histological preparation and study.

Results: Streptomycin in the larger doses employed in this series of cats causes a loss of vestibular function which becomes apparent in 8 to 10 days after the beginning of treatment: in the gait and posture, in the loss of labyrinthine reflexes, including the animals'

TABLE I.

Cat No.	Dose mg/kg	Duration of Rx (days)	Max. Vestib. Effect	Aud. Test days post Rx		Max. Microphonic response, db below 1 mv 8000	4000	2000	1000	500
1304	Control				R	--	--	--	--	--
					L	7	--	1	--	3
1323	Control				R	11	6	3.5	3.5	5
					L	--	Infected		--	--
1317	100	60	+++	18	R	11	5	2	2	3.5
					L	11	5	3.5	2	4
1209	200	32	+++	10	R	--	32	25.5	23	25.5
					L	--	19	15	14.5	17
1305	200	32	++++	22	R	37	38	40	42	42
					L	30	35	37	38	38
1314	200	52	++++	5	R	31	24	21.5	26	28
					L	32	25	22	27	28
1312	200	60	++++	15	R	14.5	10	7	8	10
					L	15.5	10	7.5	8	11
1293	100	15	+++	75	R	23	24	25	21	40
	200	26			L	--	Infected		--	--
1135	400	16	++++	60	R	--	--	--	--	--
					L	--	>40	>40	>40	>40
1310	400	32	++++	22	R	19	14	11	12	15.5
					L	31	24	21	21	24

ability to right themselves in air, and in the sudden loss of nystagmus, as is illustrated in Figs. 1 and 2. The vestibular disturbance is rated on a 4-point scale, from +, representing a just-detectable ataxia of the hind limbs to ++++, representing violent head oscillations, total loss of vestibular nystagmus in response to rotation, and ataxia so severe that the animal immediately falls when attempting to walk. All treated cats in the present series showed vestibular disturbances of +++ or ++++ severity, while the two controls showed normal reactions at all times.

When the cochlea is affected by streptomycin, it is the maximum amplitude of the microphonic response which shows the greatest change, as pointed out in the earlier publication. All frequencies from 500 to 8000 cps are affected more or less equally; hence the maximum amplitude of the response at a single frequency, e.g. 2000 cps (expressed in decibels below 1 millivolt at the round window) can be taken as a measure of the microphonic response. The average maximum response of 21 normal control ears at this frequency is 3 db below 1 mv, with a range of 1 to 7 db below 1 mv (Figs. 3 and 4).

As first shown by Davis and Saul (1934), the response recorded at the round window consists not only of the microphonic potentials, but also of action potentials from the auditory nerve. These can be distinguished most clearly in the response to a click stimulus. Fig. 5 illustrates the growth of the microphonic and nerve potentials in a normal ear as the intensity of the stimulus is increased. In cat 1305, which was severely affected by streptomycin, the microphonic response to clicks was almost completely abolished, and only a small action potential component appeared, even at very high intensities. As shown in Fig. 6, however, responses could still be recorded from the auditory area of the cerebral cortex, indicating that the central pathways must be intact.

Data for all of the animals in the present series are summarized in Table I.

PATHOLOGY-VESTIBULAR APPARATUS

The chief interest was primarily in the crista of the horizontal (lateral) canal. There are several reasons for this: 1. Sections of the cat's ear were cut at an angle which made study of the horizontal canal simpler than the others. The superior and posterior canals

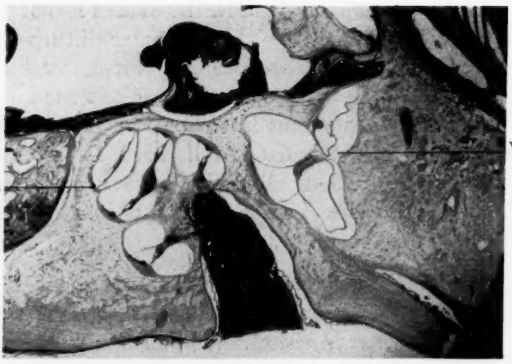

Fig. 7.—Cat 1305. X12. Temporal bone. Showing horizontal plane used in cutting several sections. This plane gives the best all around results for study of the semicircular canals, cochlea, and vestibule.

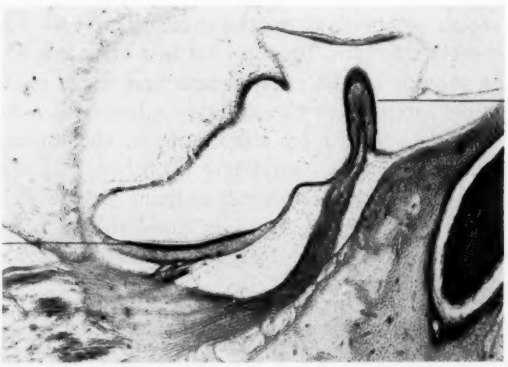

Fig. 8.—Cat 1304. Rt. X54. Normal macula of the utricle. Normal crista of the horizontal semicircular canal. Note the nerve supply of the crista.

Fig. 9.—Cat 1209. Rt. X83. Macula of utricle. Crista of horizontal semicircular canal. Note the shrinking of the macula of the utricle and the neuroepithelium of the crista. This animal received Streptomycin, 32 doses of 200 mgms per kilo., daily.

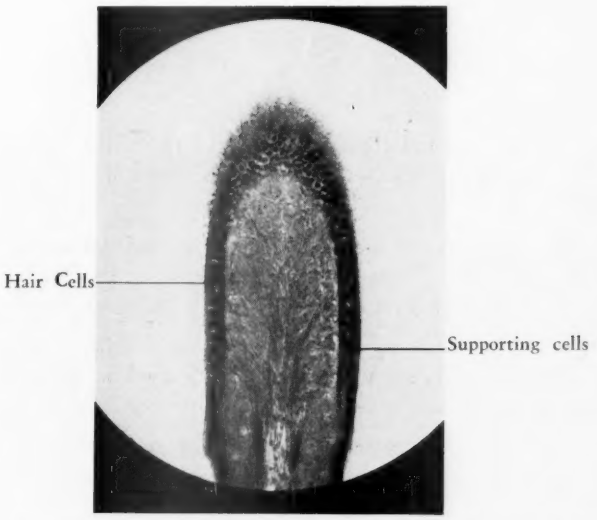

Fig. 10.—Cat 1304. X280. High power of a normal crista of the semicircular canal. Note the thickness of the neuroepithelium. The distinct row of hair cells present.

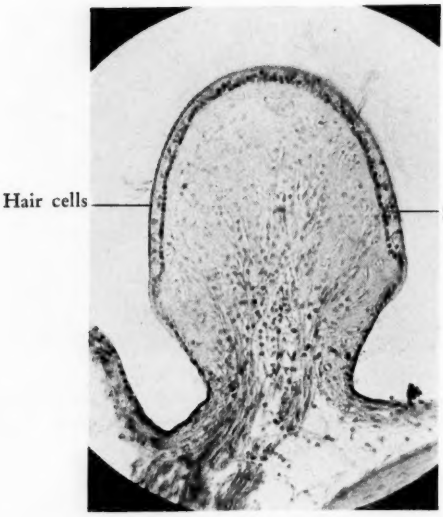

Fig. 11.—Cat 1314. Left. Crista semicircular canal X310. Note the degeneration of the hair cells. The supporting cells at the basement membrane still present and atrophied. Animal received 52 doses of Streptomycin, 200 mgms per kilo., daily.

were also studied and confirmed the findings of the horizontal canal. 2. The animal had the horizontal canals tested chiefly on the tuning table.

The crista of the horizontal canals showed definite degeneration of the hair cells. There was also shrinking of the epithelium. This was not due to fixation as normals under the same condition showed no shrinking (Figs. 9, 11, 12, 13).

The hair cells were definitely degenerated or absorbed with the supporting cells still present at the basement membrane.

We believe the position of the capula or its disappearance is not indicative of any pathology as it also occurs in sections of normal crista.

Nerve staining was not done. Scarpa's ganglia was not studied with any special nerve ganglia cell stain.

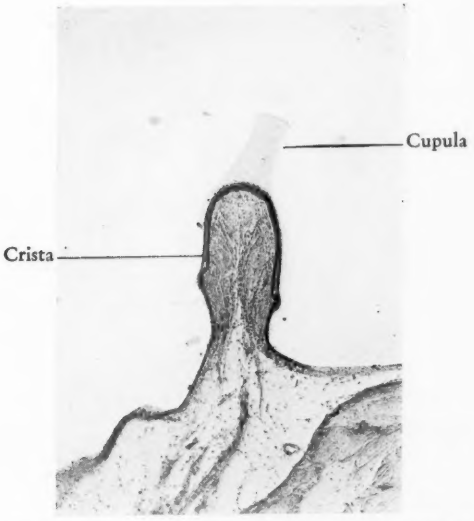

Fig. 12.—Cat 1312. X140. Cupula and crista of semicircular canal. Cupula in normal dilatation and appearance. Under this power no changes of the neuroepithelium can be made out. Streptomycin dosage, 60 doses of 200 mgms per kilo., daily.

The utricle changes were harder to evaluate, unless the neuro-epithelium was cut at right angles. In most of the sections it was cut at an angle or a tangent and, therefore, it was difficult to evaluate the degeneration present.

In the cases where the macula of the utricle were cut at right angles, there was definite shrinking of the epithelium and scattered degeneration of hair cells, but in all cases there were hair cells present and hairs projecting from them (Fig. 9).

The saccule was also difficult to evaluate for the same reason. All those studied showed practically normal neuro-epithelium, and few changes in the neuro-epithelium.

The vestibular disturbance appears fairly early by the rotation tests and if the animal is sacrificed too early, little or no changes may be found in the crista of the canals for the degeneration has not had time enough to take place.

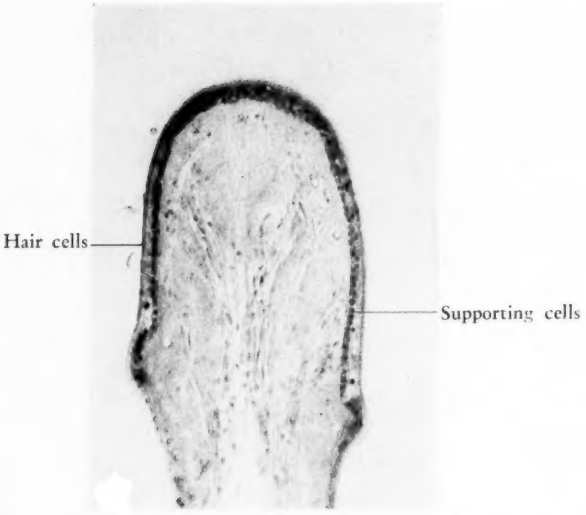

Fig. 13.—Cat 1312. X400. High power of crista shown in Fig. 6. Degeneration of the neuroepithelium supporting cells present at basement membrane.

THE COCHLEA CHANGES

The pathological changes found in the Organ of Corti are similar to those reported in previous articles on acoustic trauma, toxic conditions from infection or drugs, and inherited deafness. The changes present ranged from degeneration of the internal and external hair cells to complete absorption of the Organ of Corti.

The spiral ganglia showed in some cases almost complete degeneration and in others partial or no degeneration.

The stria vascularis showed little variation from normal except in cases with complete degeneration of the hair cells, and spiral ganglia cells where there was atrophy of the stria vascularis.

In some of the cochlea examined a degeneration of the Organ of Corti was found which differs from the type we have found in animals with degeneration secondary to acoustic trauma, inherited deafness or toxic deafness. The degeneration ordinarily found is of

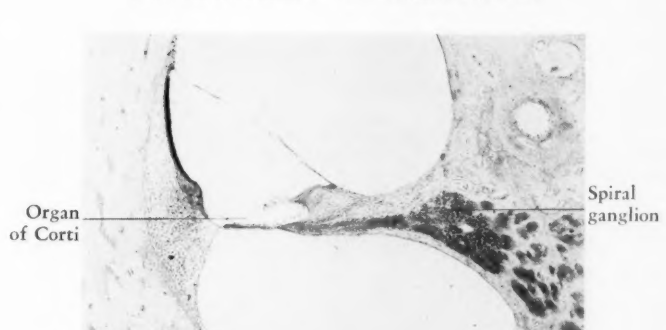

Fig. 14.—Cat 1135. Left cochlea. First turn. Left. X83. Complete degeneration of the Organ of Corti. Tectorial membrane normal and in its normal position. Note there is no evidence of degeneration of the ganglia cells in the spiral ganglion. Animal received Streptomycin, 16 doses of 400 mgms per kilo., daily.

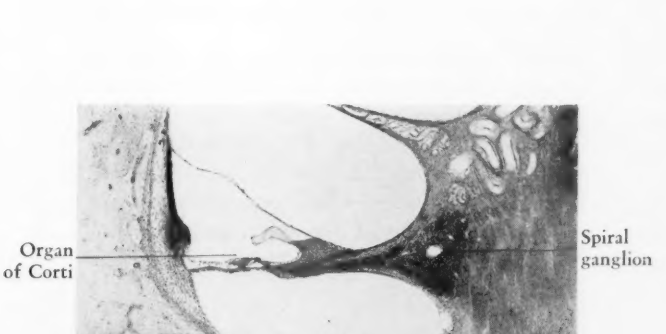

Fig. 15.—Cat 1209. Cochlea. Second turn. Right ear. X83. Under this power Organ of Corti appears normal. Nerve and spiral ganglion appear normal. Animal received Streptomycin, 30 doses of 200 mgms per kilo., daily.

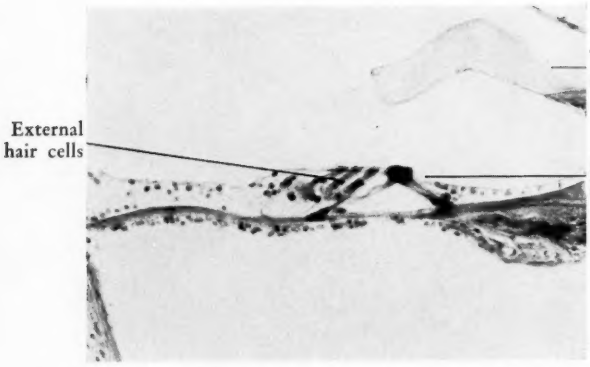

Fig. 16.—Cat 1209. Cochlea. Second turn. Right. X400. High power of turn shown in Fig. 9. Absent internal hair cells. External hair cells are present and appear normal.

external hair cells absent with internal hair cells present or both degenerated, but in these animals the degeneration was of a type not seen by before in all of our studies of the Organ of Corti.

The internal hair cells were degenerated and the external hair cells were present. There were also sections in which, with the internal hair cells gone, there was a scattered degeneration of external hair cells present. This condition was chiefly found in the upper turns of the cochlea, and the 2nd and 3rd turns; the 1st turn often having both internal and external hair cells present. There was also in one animal a degeneration of the spiral ganglia cells of the 2nd turn with this type of pathology of the Organ of Corti (Fig. 15 and 16).

This interesting finding which we have not observed before may be evidence of a special sensitivity that the internal hair cells have to streptomycin. It may give us clues as to the functions of the internal hair cells as compared to those of the external hair cells in hearing. Studies are now being carried out with this in view.

Note: In seven of the animals the brain was removed after perfusion and sent to Dr. Ida G. Schmidt, Department of Anatomy, University of Cincinnati, for his histopathological study of possible central lesions. Dr. Schmidt's essentially negative findings will be reported in a separate paper.

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DISCUSSION

Dr. J. Winston: The subject of where streptomycin and the streptomycin group of drugs attacks the vestibular apparatus—incidentally my discussion is confined to the vestibular part of the paper—is a very important question from the standpoint as to whether we are to use these drugs in the treatment of Meniere's disease. If these drugs affect the vestibular end organ only as Dr. Hawkins and Dr. Lurie have shown us, then, of course, it would be a very suitable means of treating Meniere's disease. However, my own work—and when I say "my work" I mean my associates along with me—has shown that there are definite changes in the central nervous system. Dr. Hawkins has referred to us in his paper.

A large series of cats to each of whom 14 grams of streptomycin were administered, one gram per day for 14 days, were separated into three groups. One group was vitally stained with Trypan Blue. In the second group sections of the brain and cord were stained and examined microscopically. The late Dr. Frederick Lewey, a neuropathologist of international repute, did the neuropathology. The third group was examined by surgically destroying the vestibular nuclei on the right side of the brain stem and then noting if any action from the opposite vestibular nuclei in the brain stem took place.

I want to show you some slides of our sections for the Trypan Blue vitally stained animals and those that were studied by straight histopathologic methods. The Trypan Blue was injected in the right carotid artery, in the direction of the heart because of the anatomical arrangement of the blood vessels in the neck of the cat. The innominate artery in the cat gives off the right and left common carotid arteries. The right subclavian comes off of the innominate artery and from the right subclavian artery the right vertebral artery arises. On the left side the vertebral artery arises directly from the arch of the aorta. When the dye is injected into the right common carotid in the direction of the heart, a high concentration of the dye is carried into the innominate and right subclavian artery and up through the vertebral artery directly to the base of the brain. It had been unfortunately necessary for us to ligate the common carotid artery in all of our animals, controls as well as streptomycin treated cats. The reason for this was that when the needle was inserted into the carotid artery and the dye injected and the needle withdrawn we ran into bleeding, which was uncontrollable, so that it became imperative that we ligate the right common carotid artery.

Now the question has been raised in some quarters that this may produce an artefact. Skoog has shown that when you inject a colored dye emulsion into the right carotid artery of the guinea pig in the direction of the heart and the anatomical arrangement of the blood vessels is the same in the guinea pig as in the cat, this colored dye emulsion will predominantly be seen in the right cerebellum and brain stem, whereas if the dye is injected in the direction towards the head, then it will be seen in the right cerebral hemisphere predominantly.

Boutan, investigating the site of the blood brain barrier, injected into the left common carotid artery room air in the direction of the head and produced

by the embolus a left hemiplegia in the dogs. He then vitally stained these animals and the Trypan Blue stained the cells of the left cerebral cortex as well as the adjacent areas, so that from this and Skoog's experiments we can see that by tying off the right common carotid artery, if any change in the brain is produced this change should be more or less confined to the right cerebral hemisphere, not the cerebellum and brain stem. Actually our vital staining, as you will see, has been confined to the cerebellum and the brain stem.

In conclusion I want to say that our studies have shown that the pathology was largely confined to the vermis of the cerebellum, predominating in the flocculus and nodulus, the vestibular nuclei of the brain stem, and the ventral cochlear nuclei of the brain stem.

DR. ARAM GLORIG: Many of you know that clinically I have been pursuing this lesion for about five years. Back in 1946, when Dr. Fowler and I first started we examined about 200 cases clinically from the vestibular standpoint by doing all sorts of tests, including the tilt table test, the galvanic, calorics, turning and the so-called mattress test that we discovered, and our only conclusion was that we must have a peripheral lesion of some kind. We based this mostly on the fact that with the tilt table reaction we obtained typical reactions which were caused by end-organ stimulation. When we did the galvanic stimulus we found a good nystagmus from galvanic stimulus, which we interpreted to mean that the central nuclei and the tracts from the end-organ were intact, since the galvanic is supposed to stimulate the central nervous system directly.

I then began to read these different studies that came from Winston's and Stevenson's group, and from several of the men in Europe. I began to think that perhaps our original conclusions were wrong, so I went back and reviewed our work over again. Mind you, this work was based on some 12 to 1800 calorigrams and a study of some 200 patients. When dihydrostreptomycin came along, we decided to study the dihydrostreptomycin results on the vestibular portion of the 8th nerve and we found very little destruction as far as the vestibular portion was concerned. We found that these patients began to lose their hearing which we were not expecting. Our first indication that they were losing hearing was their difficulty in talking to us and our trouble in communicating with them. We started doing audiograms and we have done about as many as 2500 audiograms on some 300 patients. From the type of audiograms which we obtained I came to the same conclusion, this must be an end-organ lesion. It started at the 8,000 area and gradually progressed towards the left. I could not conclude that any lesion could do this in so methodical a manner.

Then when Caussé, Rüedi and Hawkins began to talk about end-organ lesion, where the microphonic and action potential were affected, I began to think we are right again. Now with the microscopic evidence that we have seen today, I think our original conclusion was right.

I do not infer from this, however, that central lesions are lacking. I have never said that in any papers I have written, except that there must be a peripheral lesion and perhaps there is a central lesion, but we need further work in order to corroborate it.

The present studies that are going on in our cases show that something which Dr. Hawkins has been secretly hoping for, and perhaps we are right is that, most of the destruction in this process is due to impurities in the drug. In some of his studies with the cat he found that the purer the drug the less the destruction as far as the cochlear endings were concerned.

We have recently been running a series of cases of crystalline dihydrostreptomycin. These patients have been on 2 to 3 grams a day for about two months and so far we have found no deafness, no depression of the threshold at any frequency. We have also recently received a report from the sanitarium in Connecticut where I first started to work with Dr. Fowler and Dr. Kirby Howlett. It reports the same thing with crystalline dihydrostreptomycin. So perhaps if this is correct, we will be able to use dihydrostreptomycin and get the results we wish in tuberculosis without any damage to the vestibular or cochlear portion of the 8th nerve.

DR. STACY R. GUILD: For the first time I am convinced that the essential lesion caused by streptomycin is peripheral. I was not convinced by Caussé's report, by Berg's paper, by Ruedi's paper at our meeting last year in Atlantic City. All left me unconvinced. But here we have a very careful clinical study followed by the histologic demonstration of unquestioned changes.

Now I want to raise the question, is the action of the drug primarily on the sensory cells of the crista or is this another example of our old friend, vascular changes? From conversation late last evening the authors know the point that I make. Adjacent to the ciliated sensory epithelium of the crista is a zone of cuboidal epithelium, under which are several capillaries. Normally the structures of this region of the crista are sharply defined and they show up clearly in pictures. The region of the cuboidal epithelium is not clearly portrayed in any of the photomicrographs of the material of Drs. Hawkins and Lurie. It seems improbable that all the dozen or more sections photographed were not flat in this region and therefore were out of focus in the picture. Some of the endolymph for the vestibular part of the labyrinth is secreted by this area near the sensory epithelium of the crista. A functional disturbance of the secretory area, caused by toxic injury to the underlying capillaries, could account for the appearance of the photomicrographs. I hope the authors will examine the actual sections from the viewpoint of vascular injury as possibly being the primary lesion.

DR. JOSEPH E. HAWKINS: There is only one question that occurs to me and that is whether the vital staining with Trypan Blue which Dr. Winston describes might not have been altered in his cats after a single dose of streptomycin, as he found it to be after treatment with approximately 300 mg per kg per day for 14 days. We have found in biochemical studies with Dr. Boxer and Dr. Jellinek that streptomycin does enter the central nervous system in small amounts, and I was wondering, if these animals were tested within a few days after the last dose of streptomycin, whether the presence of streptomycin in the nervous system itself might not alter the staining of the cells. If he had tested them at a later period might they not have shown a more normal type of vital staining?

I was glad to have Dr. Glorig bring up the problem of the clinical use of dihydrostreptomycin. There was no time to touch on that in my own paper, and of course I am delighted to hear about his results with the crystalline dihydrostreptomycin, since, as he says, they bear out a notion I have had for some time that the ototoxicity of dihydrostreptomycin might be influenced at least by the presence of impurities. However, I believe the attitude to be maintained is still one of watchful waiting with all possible fingers crossed.

Of course I appreciate very much Dr. Guild's remarks, and I think that his pointing out the possible vascular changes may be extremely important; but it does leave this interesting question still untouched, namely, the very high specificity of the various antibiotics for the cochlear or vestibular structures. We have in streptomycin a drug which hits primarily the vestibular system and in

neomycin, a close relative, although its exact chemical structure has not been established, a drug which hits primarily the cochlear system. It is a very difficult thing to explain.

DR. MOSES H. LURIE: It is not very often that you can convince Dr. Guild with photomicrographs. I wish to explain, in fact all these photomicrographs that I have presented in the past years and this time were taken by my coworker, whom you all know, Dr. Werner Mueller, one of the best photomicrographers we have.

LXX

CLINICAL MEASUREMENTS AND IMPLICATIONS OF RECRUITMENT

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AND

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In 1924 Pohlman and Kranz[12] reported that: "Decreased acuity of hearing for certain limited portions of the tone scale, as determined by minimum audible sounds, is not a criterion of deafness with respect to ordinary intensities. Once the threshold of such a range of decreased acuity is attained, it takes comparatively little additional energy to cause the deficiency to seemingly disappear quite completely." In 1928 Fowler[4] noted during an audiometer test of a patient with a normal left ear but with a right ear with a loss of approximately 40 db near 4096 cycles per second that: "Comparative loudness showed that the loudness of tones (near 4096) of from 10 to 25 sensation units above the threshold in the left (non-affected) ear appeared the same loudness as 5 sensation units above the threshold in the right (affected) ear."

In 1935 Reger[13] reported that individuals with high frequency hearing losses experienced an abnormally rapid increase in loudness when intensity level was increased above the threshold value at which the loss existed. Also that such individuals with raised auditory thresholds could detect smaller increments in intensity than the normal ear at intensity levels the same number of db above the respective thresholds of each. Beginning in 1936, Fowler[5-7] published a series of papers about this abnormally rapid increase in loudness as the intensity is raised gradually above the thresholds of patients with neural deafness, to which he applied the term "recruitment." Fowler's investigations led him to the conclusion that the presence or

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absence of recruitment constituted a clinically useful method of differentiating between conduction deafness and neural deafness.

In 1948 Dix, Hallpike and Hood[2] published a study which indicated that patients with hearing losses resulting from acoustic nerve tumors did not experience recruitment but that those with labyrinthine hydrops did experience recruitment. This finding has since been confirmed by Eby and Williams,[3] Luscher and Ermanni,[11] Lundborg[10] and others. The significance of this observation to the accuracy of otological diagnosis cannot be overestimated. Recruitment tests cannot be relied upon to differentiate between middle ear and not-middle ear pathologies, but become invaluable as an aid in distinguishing between two different general types of not-middle ear pathologies. This is a fortunate finding since previous to the time that Dix, Hallpike and Hook published their work, no functional hearing test was known which would permit a distinction between general types of not-middle ear pathologies.

It now appears highly desirable to make recruitment tests on all patients with not-middle ear deafness—especially those in whom the history and physical examination are not indicative of the etiological pathology. Also it is now time to agree on uniform terminology to designate the two general types of not-middle ear pathology, namely, the type in which neural tissue concerned with the stimulation, transmission and reception of auditory stimuli has lost its irritability and the type in which the not-nerve structures within the cochlea are involved, such as the hair cells. The term "neural" pathology or neural deafness seems appropriate to designate the one type. The writers suggest that the term "scalar" pathology or "scalar" deafness be used to designate the other type of not-middle ear pathology. The term or phrase "scalar pathology" is not charged with other meanings and implies that the pathology is limited to the not-nerve structures within the scala media, scala vestibuli or scala tympani. The term "cochlear pathology" or "cochlear deafness" is acceptable only if "cochlear pathology" is defined in such a way that the ganglion cells and other neural tissue within the cochlea are excluded.

Since the presence or absence of recruitment possesses diagnostic significance in cases of not-middle ear pathology, especially in cases in which the history and physical examination are not conclusive, an

obvious question arises. How can the presence or absence of recruitment be determined?

Several different tests are being employed for the measurement of recruitment, some of which have limited application. The alternate unilateral equal loudness level balancing test is limited to unilateral impairments. The equal loudness level contour test is limited to individuals with greater impairments over part of the audible frequency range than the remainder of this range. The two preceding tests are of no value in the measurement of recruitment in individuals with equal flat bilateral hearing impairments. Intensity difference limen tests have a wider application since they can be employed with any type of unilateral or bilateral loss. Bekesy[1] has described a new type of self-testing, automatic recording, continuously variable frequency audiometer which records graphically the intensity range over which the test tones must be increased and decreased to be perceived and not perceived. This intensity range is significantly reduced in cases with scalar pathology. The Bekesy test also can be used with any type of unilateral or bilateral hearing impairment. Other types of recruitment tests, including differential masking of pure tones and speech function or speech intelligibility curves, also are in use. It should not be overlooked that appropriate masking must be employed over the ear with the better acuity while making any type of threshold or above threshold measurement on unilateral loss cases in which the difference in acuity between the ears exceeds 40 db.

In order to study the relative effectiveness of several different types of recruitment tests a special versatile instrument was constructed specifically for this purepose, the schematic of which is shown in Figure 1. The instrument consists essentially of a modified Bekesy audiometer especially adapted to test auditory acuity at any desired single frequency level over any desired time interval. The instantly reversible motor rotates the shaft of Attenuator 2, which shaft is geared to recording pens B and C. Both Attenuator 2 and Attenuator 1 are continuously variable intensity ladder attenuators, each covering an intensity range of 65 db. A cone type reduction gear shift (not shown) between the instantly reversible motor and the attenuator shaft permits instantaneous selection of any one of four different desired attenuation rates—2, 1, 0.5, or 0.25 db/sec. The direction of rotation of the armature of the instantly reversible

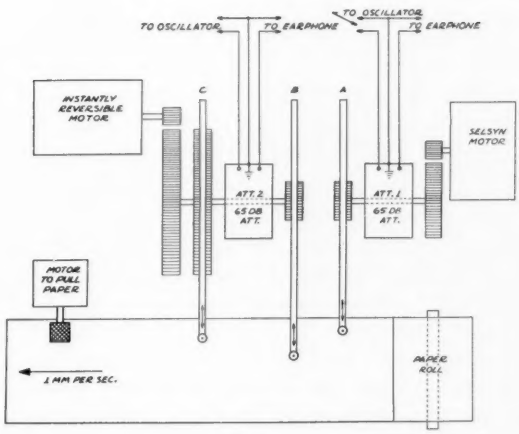

FIG. 1

motor (and indirectly the direction of the rotation of the attenuator shaft) is determined by a key which the patient operates during the test. The patient is instructed to press the key whenever and as long as he can hear the test tone in an earphone. When the tone can no longer be heard the key is to be released until the tone can just be heard, whereupon the key is to be pressed until the tone just barely becomes inaudible, and so on throughout the test. As the shaft is rotated clockwise and counter-clockwise the recording pens move to and fro, tracing a zig-zag line on the paper roll (6 inches wide) as it is pulled from right to left with a velocity of 1 mm/sec. by a friction roller attached to the shaft of a constant speed synchronous motor. As the output intensity of the attenuators increases the pens are propelled away from the attenuator shafts. Pen C is geared to move with four times the velocity of pen B to amplify the narrow intensity excursions produced by patients with marked recruitment. Only Attenuator 2 and pens B and C are used while obtaining threshold recruitment data.

In order to facilitate measures of alternate unilateral equal loudness level balances, equal loudness level contours and simultaneous bilateral equal loudness level balances, a second attenuator, Attenuator 1, driven by one of a pair of Selsyn motors, was placed near

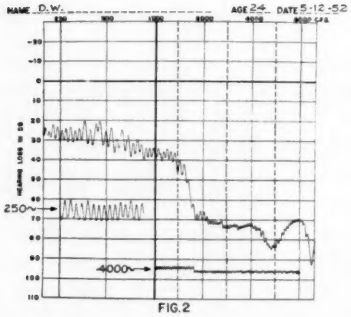

FIG. 2

Attenuator 2. The shafts of the two attenuators are in line and the racks to which the pens are attached are paralled with each other. The second Selsyn motor is in the sound proof room with the patient, who is instructed to rotate a knob on the shaft of his Selsyn in the clockwise direction until he can just barely hear the test tone and then in the counterclockwise direction until the tone just disappears, and to continue this alternate clockwise and counterclockwise rotation until he is ready to make a judgment or decision. (Selsyn motors are used for remote control of movement and force. If the shaft of one of a pair of Selsyns is rotated clockwise 15 degrees, for example, the shaft of the other executes an identical rotation.)

This combination of two attenuators, two earphones and two different sound sources, in conjunction with electronic switches and mixer amplifiers, permits an exceedingly flexible tone presentation sequence, in which the same or two different frequencies can be presented simultaneously or alternately in the two ears or two different frequencies can be presented alternately in a single ear, the loudnesses of which are under the control of either the experimenter or the patient as desired. The added combination of the electronic switches and mixing amplifiers also facilitates the use of masking when indicated in the ear opposite the ear under test.

Due to the interest of the writers in Bekesy audiometry, the acuity of most subjects is tested first on a regular continuous frequency Bekesy audiometer. Figure 2 shows such a test on one ear of an individual whose only known etiology was measles at three

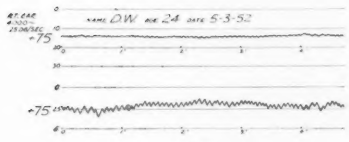

FIG. 3

years of age. The amplitude of the excursions to the right of the arrow near 250 cycles per second shows the intensity range at this frequency level, which is approximately 8 db. The amplitude of the excursions to the right of the arrow near 4000 cycles per second shows the intensity range at this fixed frequency level, which is approximately 2 db. This narrow excursion indicated the presence of recruitment at this frequency level in the patient whose audiogram is shown. (The apparent intensity levels at which the amplitudes of the excursions were obtained at the two different indicated fixed frequency levels are to be disregarded, since the input levels to the attenuator were varied in order that the excursions be recorded on "clear" parts of the audiogram. Inspection of the top threshold acuity curve indicates the hearing loss to be approximately 28 db at 250 cycles per second and 73 db at 4000 cycles per second. The attenuator of the Bekesy audiometer on which this audiogram was obtained attenuates over a 140 db range in two db steps.)

Figure 3 shows this same patient's intensity excursion at threshold at 4000 cycles per second on the Bekesy modification of Figure 1. The top excursion shows an intensity range of approximately 1 db or slightly less. (The upper horizontal parallel lines opposite the 0, 10, 20, 30 and so on show the intensity Y-axis or ordinate of pen B in db and the lower three parallel lines show the intensity Y-axis or ordinate of pen C, which is four times greater than that of B.) The legend to the left of the upper excursion made by pen B indicates that the test was made on the right ear at 4000 cycles per second with an attenuation rate of 0.25 db per second. The +75 indicates that the patient had an approximate 75 db loss for the indicated frequency. The numbers along the parallel lines indicate elapsed time in minutes. The lower three parallel lines show the amplitude of the excursions of the "amplifier" pen. Note that the db value between adjacent lines is 2.5 db whenever the "amplifier" pen is used.

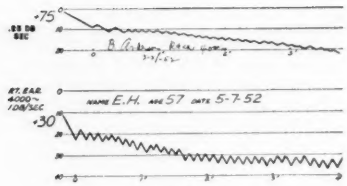

FIG. 4

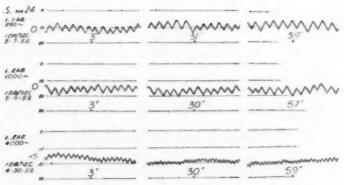

FIG. 5

Since pens B and C cannot be arranged to travel in the same straight Y-axis, the traces made by the two pens are displaced by a constant distance factor.

Figure 4 shows the amplitude of the excursions of two different individuals at 4000 cycles per second. The upper tracing indicates more recruitment than the lower one. But the most significant thing about these two tracings is the slope of each of them, indicating that a progressively higher intensity level is required to produce an audible tone the longer the patient listens to the tone. The upper tracing indicates a threshold shift of about 10 db in three minutes, the lower a shift of slightly more than 10 db in two minutes. It seems preferable to call this variation in acuity a temporary threshold shift (TTS) rather than auditory fatigue at the present time.

Figure 5 shows the relatively slight TTS of a university student with normal hearing who was tested for one hour at each of three different frequency levels. The upper curves show the acuity at 250 cycles during the second and third minutes, the 30th and 31st and the 58th and 59th minutes. The acuity remained relatively

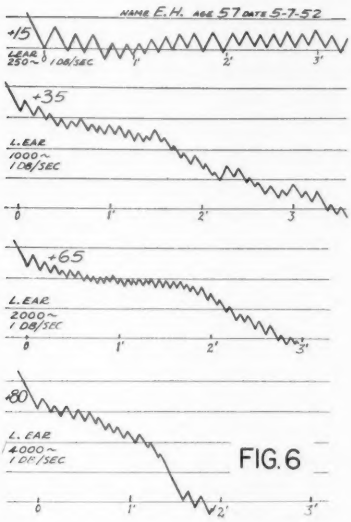

FIG. 6

constant at both 250 and 1000 cycles per second over the one hour testing time. At 4000 cycles per second here was a TTS shift of about 5 db at the end of the hour. However, most of this shift had occurred by the end of the 4th minute of testing time. These data on this individual suggest that the shifts in threshold acuity shown in Figure 4 are real and not due to an artifact of some type.

Figure 6 shows the TTS of a patient who had an acoustic nerve tumor (later confirmed by surgery). The patient had a 15 db loss at 250 cycles per second and did not demonstrate a shift over a three minute testing interval; at 1000 cycles per second, where the patient had a 35 db loss, a TTS about 25 db occurred in three minutes; at 2000 cycles per second, where the patient had a 65 db loss, a TTS of 25 db occurred in three minutes; and at 4000 cycles per second, where the patient had a loss of 80 db, a TTS of 30 db occurred in slightly less than two minutes. This patient's acuity was retested as above the following day with strikingly similar results. (The hearing losses indicated at the different frequency levels were obtained with clinical and continuous frequency Bekesy audiometers.) An 80 db level (above the threshold of the normal ear) thermal mask-

ing noise was used over this patient's left ear while obtaining the measures at 2000 and 4000 cycles per second on the pathological ear.

At first it was difficult to believe that threshold shifts of such magnitude could be produced within such short intervals of time. The equipment and procedure were checked and double checked for some type of systematic error. Some hard of hearing individuals with extreme losses do not show any appreciable threshold shift within five minute testing intervals. Threshold shifts comparable to those shown in Figure 6 have been observed in several patients. Not all individuals with recruitment exhibit a threshold shift. Nor do all not-middle ear impairments experience threshold shifts. More work is needed to learn the exact significance of the shift.

A search of the literature revealed two earlier studies concerning the TTS which are of note. Huizing[9] had made a comparison of auditory fatigue in cases with and without recruitment. Huizing, however, stimulated the ear over a period of three minutes at 2000 cps and at an intensity level of 30 db above the pathological threshold, after which the threshold measurements were again obtained. Huizing found that in cases of inner ear deafness without recruitment the shift was 5-11 db whereas the cases with recruitment showed a threshold shift of between 19 and 22 db.

An exhaustive paper on auditory fatigue resulting from threshold stimulation published by K. Schubert[14] in Germany in 1944 is just reaching subscribers in this country at the present time. Our findings agree substantially with those reported by Schubert . We have found greater shifts over shorter time intervals and have observed shifts which develop slowly at first and then more and more rapidly with time—a departure from the commonly accepted logarithmic progression of threshold shifts due to noise exposure.

It is apparent that threshold shifts produced by barely audible tones must be considered in the future. As is obvious, threshold shifts contaminate all types of recruitment measures in individuals who experience such shifts. Any type of above threshold stimulation, including alternate unilateral equal loudness level balancing, equal loudness level contours, and intensity difference limen tests may produce threshold shifts which decrease the loudness of the test tones presented to the pathological ear. Attempting to measure the degree of recruitment under such conditions is analagous to trying to measure the height of a horse while it is running. Fortunately,

Bekesy audiometry presents a technique by means of which simultaneous measurement of both TTS and recruitment is possible.

The TTS further complicates an already complicated subject. Many questions concerning recruitment remain unanswered. The literature suggests that some patients have recruitment only at threshold or near threshold intensity levels. Others do not exhibit recruitment until the stimulus tone is presented at a level several db above the pathological threshold. Harris, Haines and Myers[8] have described four different slopes or types of recruitment curves.

The writers of the present paper dare suggest that the whole field of recruitment measurement needs reevaluation under carefully controlled conditions, in which the presence or absence of TTS is taken into account.

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DISCUSSIONS

DR. EDMUND P. FOWLER, SR.: This paper interests me very much. I can agree with it. It is logical and it is what we must expect. We must remember that sensation measurements do not always agree with the stimulus.

Another thing, when patients are tested over and over again and if they are alert and on the job, they sense the timing of the variations by jumping the gun and they often make their tests seem better than they actually are. You yourself if you test yourself on an audiometer can squeeze it up a little bit more than if somebody else tests you.

I never said that recruitment was necessarily present in nerve deafness. I have always been very careful to say that recruitment when it was present was pathognomonic of nerve deafness, which is quite a different statement. However, it is true it is conceivable, that blocked lesions anywhere along the auditory tract could so affect sensations that you would not get any recruitment in the presence of neural lesions.

I recently talked this over with Dr. Lorente de No. He has just been abroad where he has been through all the laboratories where they are doing these tests (much more than in this country) and he still agrees with me. The difficulty with so many of these new tests is that they are too complicated. The clinician cannot use them. We must have simple tests and after all, the simplest test of all, if there is a binaural deafness, is the facility with which a person determines the threshold. Every otologist knows that in neural deafness there is a very definite threshold. That is due to recruitment. In obstructive deafness the threshold wobbles around a bit up and down. When you use masking you are complicating your picture. You don't know how much vibration is going through the skull if you use a masking sound around 35-40 db or more, and maybe less in thin skulls. There is only an impedance of 4 or 5 db across the skull and therefore masking sounds easily will interfere with the testing in the oposite ear of course.

When I spoke to Hallpike two years ago in London about this, he said he would look into it and see what the situation was. I have not heard from him. It may be that the lack or lessened recruitment he related in neuroma of the 8th nerve was due to faulty masking.

Fatigue itself, of course, causes recruitment, because in the first place it tires the nerve and the brain. It pulls down the threshold and it causes therefore a neural deafness, so you get increased recruitment with fatigue, and is the same way if loud tinnitus is present. Also you get a recruitment from the busy fibers creating the tinnitus.

The V. Bekese audiograms are very interesting but I do not believe that more than one or two of you have his apparatus. It is very expensive. Again I say we must have simple tests for recruitment.

As far as the nerve tumors go, I have only reported two cases where the nerve tumor was removed, and they both showed recruitment. There are therefore cases of nerve tumors that have recruitment. I trust others will be able to report further on that subject.

DR. EDMUND P. FOWLER, JR.: As usual, my father is a couple of steps ahead of me. I should like to emphasize, however, that we have had three cases in the Neurological Institute where recruitment was present with cerebello-pontine angle tumors proven at operation.

I could not quite follow Dr. Reger's remarks about Dix and Hallpike. I am not quite sure if Dr. Reger agrees with them or not. For the record, I should like to state that I think the idea of Dix and Hallpike to the effect that recruitment does not occur when the lesion is in the eighth nerve trunk and that recruitment does occur when it is in the end organ is a very interesting idea, and it may be so, but I do not think their data proves it. Masking was not used in their cases. There is no proof of any nerve elements to recruit in the ears reported. As for simple methods for the study of recruitment, I agree with Dr. Reger that the ideal thing would be for all audiometers to have continuous intensity attenuation dials, but I do not think that the complicated automatic features of Bekesy's audiometer are necessary. One can manually swing back and forth across the threshold with intensity steps at one or two decibels and simply and much more cheaply get the sort of test Dr. Reger showed in the lower part of his last slide where thresholds were shown for 4000 and 250 cps. For diagnostic purposes in cases where the loudness balance can not be done such a swinging back and forth across threshold is very useful. My father has indicated that even with five decibel steps in the ordinary audiometer you can tell qualitatively the difference between nerve and conduction deafness. With one or two decibel steps this is much easier. Incidentally, Bekesy's audiometer has two decibel steps, so that one decibel steps and manual bringing back and forth by the operator would be better than Bekesy's audiometer from this standpoint. I would like to emphasize last of all, another remark of my father's that the apparent variability of thresholds in normal and conductive deafened ears. We have recently been doing threshold tests on the vestibular apparatus and find much the same thing as in the auditory labyrinth. In these experiments, the patient sits in the chair blindfolded with the motor going; he then indicates whether the chair is rotating to the right or left or standing still. One goes through very much the same mental process as when pushing the button on the Bekesy audiometer. There is the same sort of lag depending on the mental reaction. This lag is the most serious objection to my mind to the Bekesy audiometer. Unless you take highly trained (I should say Reger trained) technicians you are not likely to get consistently clear cut responses on all types of individuals. In the run of the mill clinic patients we do not know if it is the lag of the thumb or the lag of the cerebral cortex which makes the time between changes in stimulus so long. With the vestibular threshold tests the subject does not give consistent responses until he gets well over a certain acceleration threshold. He can sit perfectly still and maintain the same acceleration for a consistent period but will respond more or less rhythmically first to the right and then to the left and then perhaps say that he is "still." If I remember my physiology correctly, these vacillations at thresholds occur with sight and with touch; with smell they are even more marked and prolonged. In all cases they are much the same sort of thing. I think we must consider the reaction time of a given individual very seriously in this type of testing unless we are testing highly trained individuals.

Lastly, I hope my remarks will not discourage any of you or Dr. Reger from continuing this line of investigation which, to me, is most promising. We are

certainly not going to get anywhere if we let minor objections to experiments discourage us. Until we have more and better tests for quantitatively differentiating the various types of deafness we will continue to be confusd by many of our cases.

DR. KINSEY M. SIMONTON: We have used the binaural loudness balance test on a great many of our patients who present the diagnostic problems of vertigo and unilateral deafness, and have concluded that the test is a valuable aid in differentiation between lesions of the cochlear end-organ and those of the acoustic nerve tract. The results of the binaural loudness balance test have correlated well with the other symptoms of the labyrinthosis syndrome, outlined by Mygind namely, that the end organ lesion presents deafness predominantly of the low tones, that the threshold of pain is reduced and that the discrimination for speech is decreased. Study of this data revealed a dividing point in the degree of recruitment necessary to make the clinical distinction between lesions of the end-organ and lesions of the nerve. Low degrees of recruitment in which the equal intensities never give equal loudness sensation may be found accompanying the neural lesion. Patients who show the end-organ type of lesion present a very high degree of recruitment as indicated by the binaural loudness balance test. Equality of loudness sensation occurs with equal intensity at around 80 db of intensity. It is our opinion that recruitment of loudness measured by the binaural loudness balance test gives information of value in making the differentiation between lesions of the cochlea and those of the end-organ.

DR. SCOTT REGER: I wish to thank Dr. Fowler very much for being so kind to me and with his permission, I shall insert the phrase that he quoted, namely, that "recruitment is pathognomonic of nerve deafness." I am remaining absolutely open-minded about whether you find recruitment in scalar deafness. I see smiles and the reaction time was not very slow.

I have seen only one patient with a clinical diagnosis of some sort of tumor of 8th nerve. In this patient recruitment was not present and fatigue was very rapid. I have seen marked fatigue in people with what I would consider marked recruitment.

I think that Dr. Sinmonton's insistance upon degrees of recruitment is important. It is not a proposition in which you have or don't have recruitment. You either have a little bit of it or a whole lot of it. Until we have procedures with which we can assess the meaning of little or large recruitment we are not going to arrive at the understanding we hope to attain. Neither are we going to arrive at the understanding of this phenomenon which we hope to attain by testing clinically with a 5 db step audiometer and observing the patient's reaction. I agree that one can tell if there is recruitment of a pronounced degree or not. At the present time Dr. Fowler's suggestion is practical and useful but I am attempting to pin this thing down. When I built this complicated gadget I showed you I was going to do Békésy audiometry; do a binaural altered loudness balancing. I attempted it on one patient who fatigued so quickly for the tone presented to the pathological ear I just could not get any measurement. It was apparent I would not get anywhere so this is what I am doing from now on: I am testing the people on the Békésy threshold for five or ten minutes. If they do not develop fatigue, then I will have them come back the next day and I will make balancing, discrimination and all the other recruitment measures. I do not find very many people with a hearing loss that don't fatigue so rapidly that you cannot keep up. If you are going to have five db step attenuators you have a chance to catch up with them because it may take a few seconds to produce a

five decibel shift in threshold but your error will be of the order of magnitude of 5 db.

One remark about lag and reaction time. Remember in the young man in testing the high frequencies we have very narrow swings and in the low frequencies we have wide swings. I think we are safe in assuming we would have the same lag if the individual has one normal and one pathological ear. I suspect his reaction time or lag would be the same in the normal ear or the pathological ear. I am subjecting this to research. I am doing this this summer with my classes. I am having some students measure reaction time. I am going to take those with the slow reaction time and those with the fast reaction time and do the Békésy test on them with four different variations. I will have the intensities increased 2 db, 1 db, .5 db, and 0.1 db per second, if necessary, to measure what the differences in lag and reaction time are under carefully controlled conditions.

I am astounded by the fact that you can take the average housewife and farmer and tell them how to take the test on the Békésy audiometer and have them do a beautiful job. I am testing five to seven year old children. I tested a four year old the other day who had a swing of only 20 about the threshold as a mean, who should have had normal hearing since he had no aural pathology of any kind, but I don't recommend relying on results that I might get on a four year old child because unless you have vertification with other types of tests you may get into difficulty.

LXXI

SOUND CONDUCTION IN THE COCHLEA

ERNEST GLEN WEVER, PH.D.

AND

MERLE LAWRENCE, PH.D.

PRINCETON, N. J.

Two principal types of theory have been developed to explain the conduction of sound within the cochlea. They may be called the fluid theories and the membrane theories according to the medium in which the conduction is supposed to occur. The fluid theories have grown up primarily in connection with the resonance hypothesis, as best known in the formulations of Helmholtz and his followers. The membrane theories have developed in opposition to the resonance hypothesis and in the search for a new principle of cochlear action to take the place of resonance.

1. *The fluid theories.* According to the fluid theories the alternating pressures caused by the vibratory movements communicated to the oval window are exerted upon the fluid throughout the cochlea and pass directly from this fluid to the various parts of the basilar membrane and its associated structures. An inward thrust of the stapes produces an excess pressure in the perilymph of the scala vestibuli and because Reissner's membrane is exceedingly thin and yielding this pressure is transmitted immediately and with no appreciable decrement to the endolymph of the cochlear duct. A positive pressure is thus exerted upon the surface of the basilar membrane and as a result this membrane is displaced toward the scala tympani. An outward movement of the stapes produces a contrary displacement of the basilar membrane, and these alternating displacements in response to a series of sound waves cause a periodic

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Presented by invitation to the American Otological Society at the Toronto meeting on May 19, 1952.

The experiments described here were aided by a contract with the Office of Naval Research and by Higgins funds allotted to Princeton University.

Preliminary results of this experiment have been reported recently.[1]

stimulation of the sensory cells. The important feature of this theory is that a given portion of vibratory energy finds its way to some particular region of the basilar membrane directly by way of the cochlear fluid. A region of the membrane at the apical end of the cochlea, for example, is set in motion because a part of the vibratory energy passes through the column of fluid extending from the stapes all along the cochlear spiral to this region.

2. *The membrane theories.* The membrane theories, on the contrary, conceive of the transmission of vibratory energy through the cochlea as achieved principally by a wave motion in the basilar membrane or, sometimes, in Reissner's membrane. The movements of the stapes are transferred to the cochlear fluid just beyond the oval window and then immediately to the basal end of the basilar membrane, and thereafter the movements constitute a wave traveling up the membrane. A useful analogy, which aids us in picturing the process that is conceived here, is a rope that is attached at one end and whose other end is held in the hand. If the end of the rope is given a quick flip a transverse wave is produced in the rope and travels at a moderate speed to its farther end.

Early in the development of auditory theory the principal support was given to the fluid theory of cochlear transmission. The membrane theory received scant attention and appeared only in the relatively obscure theories of Hurst, ter Kuile, and Watt. Recently, however, this theory has enjoyed a phenomenal growth of interest and development, until now there are 12 different traveling wave theories. This new development received its primary impetus from the experimental work of Békésy in 1928.[2] In mechanical models Békésy was able to see the traveling waves as set up by sounds and from indirect evidence gained in visual observations on cadaver specimens he concluded that these waves were likewise present in the living ear. The many traveling wave theories that have appeared in recent years represent different attempts to provide descriptions, largely in mathematical terms, of the origins and forms of the sort of waves that Békésy described.

The basic principle of the traveling wave theories is that all regions of the membrane beyond the basal end receive their vibratory energy from regions of the membrane lying just below them along the cochlear spiral. The cochlear fluid plays only a secondary

role in this process of energy transmission. On this assumption the various formulations of the theory are in full agreement.

These formulations are also in agreement in their assumptions about the general form and course of the traveling wave and its essential role in stimulation of the sensory cells. There are differences in detail, but the following features are always present in some form.

(1) As an effect of some one pulse of sound a wave arises at the basal end of the basilar membrane and travels apically. Successive pulses produce a series of waves of this kind.

(2) This wave travels along the membrane, varying in amplitude as it goes. Generally speaking, the wave rises in amplitude for a way, attains a maximum at some point in its course, and then quickly dies away.

(3) The particular form of the wave varies as a function of frequency. The rate of rise in amplitude, the point in the cochlea reached at the maximum, and the region of expiration of the wave all vary systematically, with the higher tones passing through these changes the more rapidly. Thus the highest tones attain their maximums and expire at the extreme basal end of the cochlea whereas the lowest tones proceed all the way to the apex. Some feature of the wave—its maximum point or its point of expiration—is responsible either directly or indirectly for stimulation of the sensory cells.

(4) The speed of wave transmission along the cochlea is relatively slow. It is far below what would be expected for the transmission of sound waves through a free fluid. This is necessary in order that the wave motion shall be suitably represented along the length of the basilar membrane. An irreducible minimum is that just over one fourth of a wave length shall be covered on the basilar membrane, for otherwise a maximum of amplitude could not be reached. Most of the theories assume that several waves are simultaneously present. Therefore the rate of propagation must be slow relative to the period of a wave. To represent the lowest perceptible pitches, which extend to 15 cycles, the speed of wave propagation must be below 2 meters per second for a fourth of a wave and below 0.5 meters per second for a complete wave. It may be mentioned here that sound in open water at body temperature travels 1563 meters per second.

We see in the traveling wave theories that all tones in their actions are spatially distributed along the cochlea. Spatial position

is then used as the cue to pitch perception. In this respect this type of theory resembles the resonance theory that it was designed to supplant.

The specific character of the assumptions made by the traveling wave theories, as described above, makes it possible to design an experimental test of such theories. We have made a series of observations for this purpose. These experiments were carried out on anesthetized cats, with use of the cochlear potentials to indicate the action of the inner ear.

We stimulated these ears in two ways. One way was as usual, with an aerial sound introduced into the external auditory meatus and reaching the cochlea through the ossicular chain and the oval window. The other way was through a small hole drilled through the cochlear wall in the apical region. The stimulus was introduced into this hole either as aerial sound or as the vibrations of a needle inserted into the hole and driven by a crystal vibrator. Both means of apical stimulation were used for all critical tests. In aerial stimulation at the apical hole a leakage of sound to other regions was prevented by introducing the sound through a tube carefully sealed over the hole. Thorough tests were carried out to show the absence of any appreciable leakage of sound into the air and likewise the absence of any appreciable vibration communicated to the bony capsule of the cochlea.

The basic experiment consisted of stimulating the cochlea simultaneously with the same tone by basal and apical routes. For this stimulation we used a single audio-oscillator followed by a divider network that delivered the alternating currents to two channels where they could be independently controlled. Controls were provided both of stimulus intensity and of the phase relation between the two channels. The phase changer had a complete range of 360° by steps of 5.° One channel led to a loudspeaker connected through a tube to the external auditory meatus and the other channel led either to a second loudspeaker or to the crystal vibrator already mentioned.

With simultaneous stimulation at the two ends of the cochlea, the cochlear potentials were recorded with an electrode on the round window membrane. These potentials varied in magnitude according to the phase relation of the two stimuli. We may state the principle of summation of the two stimuli as follows: the resulting

potentials at all times represent a vector sum of the effects of the stimuli acting separately. This law holds strictly for all frequencies, intensities, and phase relations within the limits of overloading of the ear, and it holds for all conditions whatsoever if we take the overloading function into account.

These relations are most readily dealt with if we simplify the conditions by introducing our basal and apical stimuli separately and adjusting the stimulus intensity for some given amount of response in each case. In most of the experiments we chose the two intensities to give a response of 10 microvolts. Then simultaneous stimulation at the two ends of the cochlea gave a response that varied continuously from 20 microvolts to zero depending upon the phase relation. A certain phase relation could always be found that gave a response just double that of either stimulus by itself, and a phase relation 180° away from this one gave a cancellation of response. The phase adjustment for the null condition is much more critical than that for the doubling—this is of the nature of vector addition—and therefore we have carried out nearly all of our tests by the null procedure. It must be emphasized, however, that the same tests that are done with null adjustments can be carried out by adjustments to a maximum, or indeed to any intermediate point in the curve of summation. In other words, the rules of vectorial summation hold strictly for this kind of stimulation.

Now we need to determine where in the cochlea the summation takes place. Tests by means of an acoustic probe applied about the cochlea prove that this summation does not occur in the air. Other tests by means of a vibration pickup prove that it does not occur in the bone of the cochlear capsule.

A more elaborate procedure is necessary to test the possibility that the summation occurs in the pathways of electrical conduction of the cochlear potentials. The usual cancellation was obtained with sounds delivered to each end of the cochlea and by use of the round window electrode. Then, without any change in the conditions of stimulation, the electrode was moved to some other point on the cochlear capsule, say to the apical end. For all the low tones, up to about 1000 cycles, it was found unnecessary to make any adjustment in the stimuli to maintain the cancellation. The magnitude of response might be changed for both the basal and the apical stimuli separately, but this magnitude was changed equally for both stimuli and the

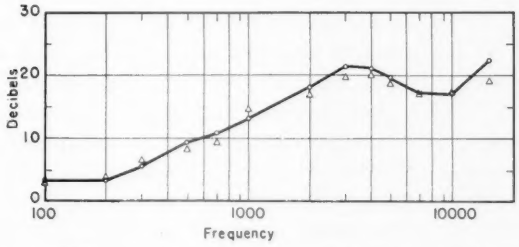

Fig. 1.—Differences, in db, in magnitudes of potentials recorded at the two ends of the cochlea, for both a basal stimulus (circular points) and an apical stimulus (triangles).

balance between their two effects remained unaltered. The previously existing phase relation also remained unaltered, at least within our ability to determine it. The electrode may be moved to any point on the cochlear capsule and this constancy holds.

When high tones are used in this situation the balance between the two stimuli is slightly disturbed and minor adjustments of intensity and phase have to be made to restore the null. The reason for these adjustments will be brought out presently.

The results for the low tones show that for them the summation of the effects of our two stimuli does not occur in the electrical pathways. If it did so, our balance would be seriously upset on moving the electrode to a new position involving different distances of conduction and different resistances of pathways. When one stimulus is used the response magnitude is always changed when the electrode is moved from round window to apex. Fig. 1 demonstrates this fact both for a basal stimulus (circular points) and for an apical stimulus (triangles). We see here that the change is about 3 db for the low tones and rises progressively to more than 20 db for the high tones.

We have now excluded (for the low tones at least) all possible sites for the summation process except the fluid pathways in the cochlea and the sensory cells themselves. We can exclude the initial portions of the fluid pathways, for they are obviously different. Also, complete cancellation of two progressive waves can occur only when these waves are going in the same direction. Our waves in the coch-

lea are going in opposite directions, and though cancellation might occur at certain points, constituting nodes in a standing wave pattern, there would be action at all other points. Moving the electrode to new positions along the cochlear capsule would necessarily reveal such points of action; and they are not found. It appears therefore that our two fluid waves come together and are equally effective only at the sensory cells themselves. We conclude that the summation occurs in these sensory cells, at least for the low tones and within the limitations of measurement in our experimental situation.

Now let us consider the high tones. We show in Fig. 2 the phase adjustments required to restore a null after establishing it for a round window electrode and then moving the electrode to the apex of the cochlea at the edge of the hole that we have drilled there. We see that these phase adjustments become larger the higher the frequency, until at 10,000 cycles they reach 40.° The necessary intensity adjustments are shown in Fig. 1 for the different frequencies as the ordinate differences in db between the circular and triangular points. These differences are small, averaging a little over 1 db.

Our explanation of these results begins with the assumption, for which there is ample evidence, that a given stimulus affects various hair cells distributed along the cochlea. A stimulus delivered to the basal end of the cochlea passes through the cochlear fluid to all these cells, encountering the more basal cells earlier and the more apical cells later, because of the time required for transmission through the fluid. Also, this stimulus is stronger as it reaches the more basal cells and is weaker as it proceeds up the cochlea. Contrariwise the apical stimulus, though it reaches the very same cells, is relatively advanced in time for the apical cells and is stronger as it reaches them. The two stimuli delivered together produce effects that differ in time and intensity, but these effects can be balanced by suitable adjustments of the stimuli. (Here the phase adjustment represents the time relationship.) If the area of distribution of the cells of which we are speaking is appreciable we may expect readjustments to be necessary on moving the electrode to a new position. The fact that such readjustments are unnecessary for the low tones signifies that for these tones the time and intensity differences for our two stimuli are very small. For the high tones they become measurable, as Fig. 2 has shown, but they are still small relative to a wave length (which is 360° in terms of phase).

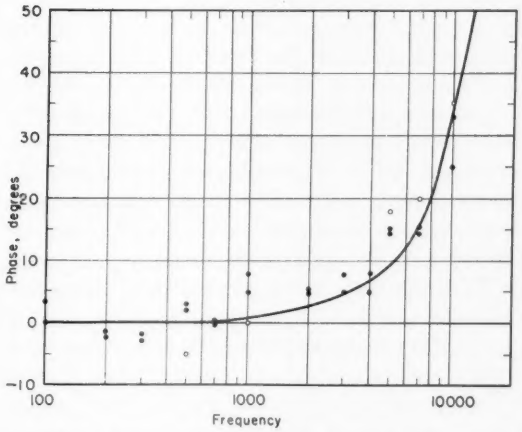

Fig. 2.—Phase adjustments required to restore the null when the electrode was moved from round window to apex.

A closer evaluation of these data requires our consideration of a further condition, which is the effect of the path of electrical conduction upon the recorded signal. We have studied this path of conduction in the cochlea by introducing electrical potentials at one end and recording them at the other end, and we find that a potential passing along the cochlea is reduced in strength by about 1 db for every mm of distance. Our two electrodes at opposite ends of the cochlea therefore give different records of the actions of different cells according to their distances from these cells. The round window electrode reports most faithfully on the basal cells and gives only a partial indication of the activity of more remote cells. The apical electrode likewise has a bias, but in the other direction. Our two electrodes thus seem to see different response regions, though they are really the same regions modified by the conditions of recording. When we stimulate at the two ends of the cochlea and adjust for a null with the electrode on the round window we are balancing an action in the more basal cells against a more vigorous action of apical cells. Then when we turn to the apical electrode we have to readjust our stimulus relations to compensate for the delay and attenuation occurring between the response regions that our electrodes seem to see. The fact that under the conditions these differences are

small signifies that the conduction times are short and the acoustic attenuation is small. The fact that for the low tones the differences are altogether negligible signifies that for these tones our two electrodes record patterns that are essentially alike. We conclude that these tones spread broadly over the cochlea and produce an action that is much the same as seen from either end of the cochlea. The high tones, on the other hand, show differences, and these differences increase with frequency. Evidently the high tones produce a response that is asymmetrical and is different as seen by the two electrodes. The results of Fig. 1 show clearly the nature of this asymmetry. The high tones have patterns of action that most strongly involve the basal end of the cochlea. The round window electrode therefore records much stronger potentials for these tones than the apical electrode does, when other conditions are kept constant. Indeed, the variations shown in Fig. 1 for the different frequencies clearly represent the increasing participation of the basal end of the cochlea as the frequency is raised.

It is possible to make a rough estimation of the speed of conduction through the cochlea on the basis of these results. If we take a curve that according to the best available data represents the form of the cochlear response curve for some tone and apply our data on the bias with which our two electrodes record this curve we shall obtain two weighted curves, one in which the amplitudes at the basal end of the true function are emphasized and another in which the amplitudes at the apical end are emphasized. The distance between the centers of area of our two weighted curves can then be found. We have made the calculations for 10,000 cycles and have obtained a distance of 6.8 mm after correcting for the spiral form of the fluid pathways. The phase difference of 40,° as shown in Fig. 2, then represents a double travel over this distance. It is a double travel because our initial adjustments compensate for a relative delay of the apical stimulus and our later adjustments compensate for a delay of the basal stimulus. Hence a distance of 6.8 mm is covered in a time represented by 20/360 of the period of a 10000 cycle wave, which is 5.56 microseconds; the speed is then 1225 meters per second. This figure is not to be taken too seriously because of the obvious uncertainty about the true form of the response wave, but we consider it to be of the correct order of magnitude. It is to be compared with the speed of sound conduction through open water, which is 1563 meters per second.

The results that we have presented here are quite out of harmony with a traveling wave theory. On a traveling wave theory it should not be possible to cancel the effects of a basal and an apical stimulus and on moving the electrode to maintain this cancellation fully, as with the low tones, or with slight variations, as with the high tones. We should have two traveling waves, one originating at the basal end and another originating at the apical end and progressing toward one another. Depending on how far these waves are presumed to go before being extinguished, the result will be two waves that never meet or two waves that overlap wholly or in part. Two separate waves would give a pattern of varying activity along the cochlea and overlapping waves would give rise to a standing wave pattern consisting of nodes and loops, and either of these patterns would readily be disclosed on moving our electrode. No such patterns are found, and hence we conclude that both the basal and the apical stimuli affect the same regions of the basilar membrane and in essentially the same manner.

This conclusion is in agreement with a local action theory. A sound applied to the cochlea spreads rapidly and extensively through the cochlear fluid and its pressure is brought to bear on all regions of the basilar membrane, and with only slight attenuation even at the distant end of the cochlea. The displacement of the membrane produced in any particular region depends upon the local characteristics of the structures in that region. This displacement and its effects in stimulation represent whatever impedances and hence whatever intensity variations and phase lags are characteristic of that region. The results reported by Békésy and others on intensity variations and phase lags within the cochlea do not require the assumption of traveling waves but are fully explainable on this local action theory.

The traveling wave theories arose largely out of a dissatisfaction with the resonance type of theory and especially a dissatisfaction as to this theory's limitations in handling the problem of pitch discrimination. In the presence of friction, which may not be denied under the conditions prevailing in the cochlea, a resonator cannot be very selective and must respond to a wide range of frequencies. The resonance principle therefore fails to account for the delicacy of our pitch perception. It was hoped that the traveling wave theory by the use of a new principle would avoid this difficulty.

It is of interest that the traveling wave idea failed even in theory in reaching this primary objective. None of the mathematical formulations has been able to predict a specific form of action for the traveling wave. Instead, this wave is represented as extending its effects over large regions of the basilar membrane. Specificity of stimulation has been brought into these theories only by the addition of special assumptions, according to which the widespread action is somehow 'sharpened up' in the cochlea or in the higher centers of the brain. These assumptions are mostly gratuitous and often unreasonable. In any case they might as easily be made for the resonance theory as for the traveling wave theory, and thus they secure no special advantages to the latter theory.

It is our view that a widespread action in the cochlea has to be accepted as a basic limitation in any theory. Except for the high tones, for which the action is somewhat specific, the spatial cue gives only a general indication of pitch. This cue therefore has to be supplemented and extended by the use of the frequency cue. As we know, this cue is available for low and intermediate tones, for these tones have their frequencies reproduced in the auditory nerve discharge. Only by the use of both cues can we explain fully the facts of pitch perception and other facts about our auditory experiences. This is the volley theory, discussed more fully elsewhere.[3]

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DISCUSSIONS

DR. STACY R. GUILD: This study pleases me especially because I have been simply swamped in attempting to understand the papers on theories of travelling waves in the cochlea. The thought that there is a 13th one in manuscript now, using the higher mathematics to explain how the ear works simply has appalled me. I certainly hope the beautiful report presented here today, very briefly, sustains the test of all attacks. I am certain there are going to be attacks.

For example, a week ago Thursday, on the 8th of May in New York City, at the meeting of the Acoustical Society, work from the Central Institute for the Deaf in St. Louis was reported by Dr. Davis and Dr. Tasaki. The observations

they have recently made provide the strongest support for the traveling-wave theory that I know about. Such data must be correlated with that reported today by Drs. Wever and Lawrence. Observations are one thing, interpretations are another.

The combination of sound in the cochlea, if the observations of Wever and Lawrence stand the test of time, becomes a matter simple to understand. Even we non-mathematical fellows, rusty even in arithmetic, should be able to comprehend this aspect of cochlear physiology.

In conclusion I would like to remark that at the meeting of this Society under the presidency of the Guest of Honor for the present occasion, namely, Dr. Fowler, 15years ago, in 1937, a symposium which lasted a day or a day and one-half was held on the neural mechanism of hearing. In it, I was asked to make some remarks on the anatomical and physiological aspects. My summary of the situation then was that in spite of terrific numbers of theories and observations, and so forth, we still do not know how we hear; and I capitalized and underscored and italicized "know." In spite of 15 years more of very active work by many investigators, I still feel the same way. We do not yet know how we hear.

DR. ERNEST G. WEVER: I am glad that Dr. Guild has referred to the mathematical complexities of the traveling wave theories. It is indeed a relief to find that the actual processes in the ear are far less elaborate than some of the theories have tried to represent them.

Let me make a remark or two about the results that Dr. Guild has mentioned, the results recently reported by Dr. Hallowell Davis and his associates at St. Louis. These investigators found that responses from different regions of the basilar membrane may sometimes show large phase differences, differences much exceeding those that we have found. They obtained differences up to 900 degrees in comparison with our maximum values of 40 degrees. This disagreement of course has to be explained.

My suggestion is that our method represents a general or average difference between large regions of the basilar membrane whereas theirs represents differences between certain specific regions. Their large differences evidently obtain between fringe regions of the responses, and it appears from our results that these fringe regions make only a slight contribution to the response as a whole.

Let me carry the problem a little further. It is true that phase differences along the basilar membrane can be accounted for on a traveling wave theory, but it is just as true that such differences do not require such a theory. An alternative explanation is that parts of the basilar membrane when stimulated by sound respond strictly according to their local physical characteristics. If those characteristics differ in different regions of the membrane the regional responses will show different time or phase lags as well as different amplitudes.

There will be objections to this explanation, I am sure, for it will be said that there are strict limitations on the amount of time lag that can arise in a resonating system or in any but a traveling wave system. However, let me point out that these expected limitations are themselves only theoretical and may not hold for the ear. At any rate, if we are to accept all the data now at hand we must seek an explanation of the phase lags without reference to traveling waves.

LXXII

ABNORMAL PNEUMATIZATION OF THE TEMPORAL BONE

LAWRENCE R. BOIES, M.D.

AND

L. IAN YOUNGER, M.D.

Today, with the changed character of otological practice, most otologists seem to be occupied with but two forms of middle ear disease. One has to do with the presence of a sterile fluid in the middle ear, the other with the matter of a chronic middle ear suppuration or its sequelae. The former seems to be on the increase. The latter is still common. One hears the query made, "If acute suppurative otitis media is disappearing, will we eventually run out of chronic ears?"

Sterile fluid in the middle ear apparently occurs from two causes. One is an effusion of serum into the middle ear and its related cavities following a blockage of the Eustachian tube. Infection, allergic swelling and sudden changes of atmospheric pressure may be etiologic factors. The other cause apparently is the aftermath of a middle ear inflammation, the exudate of which is rendered sterile by natural processes or through the effect of an antibiotic. We suspect at the present time that the apparent increase in incidence of residual fluid in the middle ear is the result of the current management of middle ear inflammation which is largely in the hands of the pediatrician or the family doctor. They have but one plan of therapy—the use of antibiotics and ear drops. Surgical drainage, which would promptly aid resolution, is no longer considered.

Informal discussions among otologists seem to lead to the conclusion that this present day treatment of middle ear inflammation is prone to produce a higher incidence of mild to moderate degrees of permanent conductive hearing loss and an increased susceptibility to inflammation on the part of one whose middle ear mucosa has been previously damaged. Also, that this present day treatment may be a factor in the genesis of chronic suppurative otitis media.

Based on personal clinical observations, this first consideration would seem to be acceptable. However, the genesis of chronic ears because of dependence on antibiotic therapy and lack of surgical drainage in acute conditions would seem to need some proof.

Let us re-examine the pathogenesis of chronic suppuration of the middle ear as we knew it prior to the era of antibiotics. Chronic ears seemed to develop under four circumstances as follows:

1. *As the sequela of a severe infection causing necrotic change in some portion of the tympanum.* This was not uncommon when the middle ear complication occurred in one of the exanthemata such as scarlet fever or measles. The damage in some cases was so extensive that ordinary repair by natural processes did not occur, and under these circumstances the ear was destined to chronicity from the start. Granulations, polypi and a marginal perforation allowing the ingrowth of squamous epithelium often became part of the picture, and eventually in some cases the development of cholesteatoma. Today, most of the chronic ears in adults in which there is a cholesteatoma occupying the antrum, or cases of chronicity with large central perforations with or without granulomata or polypi, dating from childhood, will usually give a history of an attack of scarlet fever or measles preceding the onset of the discharge. That source of chronic suppuration will practically be gone in time because the incidence of scarlet fever and measles is decreasing and the possibility of complications when these diseases do occur is less with modern antibiotic therapy.

2. *In an acute otitis media in an ear in which the mucosa is abnormal.* The behavior of an acute otitis media in an ear in which there has been very limited or absent pneumatization has always seemed different than in the well pneumatized case. The mucosa of the middle ear and antrum is hyperplastic. An acute infection heals poorly in this kind of soil. Granulations and polypi are common, but unless the perforation is marginal, the formation of cholesteatoma is uncommon. Probably the incidence of this type will be lessened through antibiotic therapy.

3. *As the result of the formation of a cholesteatoma in the attic, without pre-existing perforation.* This apparently can occur under two sets of circumstances. One is that an ingrowth of epithelium results from a retraction of Shrapnell's membrane from a

prolonged negative pressure in the attic. This negative pressure may result from the closing off of the attic by the presence of persistent hyperplastic subepithelial connective tissue sealing off the attic from the rest of the middle ear or it may result from prolonged occlusion of the eustachian tube. When Shrapnell's membrane invaginates, a blind pouch is formed. The neck of the pouch is too constricted to allow the escape of desquamating epithelium. Thus, a cholesteatoma forms. Its presence becomes known when it becomes large enough to extend out of the attic, or when infection occurs and discharge appears through a small perforation at the site the invagination started.

The other circumstances under which these attic cholesteatoma might develop is from metaplastic changes in epithelial situations at this site. Tumarkin is an exponent of this theory. That idea will be referred to later.

This type of chronic ear develops only in poorly pneumatized temporal bones.

4. *As a complication of ordinary acute otitis media in an ear with well developed pneumatization.* Ordinary acute otitis media in a well pneumatized temporal bone rarely became chronic even before the use of sulfonamides or antibiotics. The ear healed by natural methods, or surgical intervention produced a cure or the patient died of complications such as meningitis, the sequelae of blood stream infection, etc. Today, it is probable that a case of ordinary otitis media in a well pneumatized temporal bone treated skillfully with the antibiotics and surgical drainage, when indicated, never becomes a chronic.

From these considerations, it seems probable that the genesis of chronic suppurations in the present day rests largely upon the matter of limited or absent pneumatization.

What is the significance of limited or absent pneumatization? Is it correct to call it abnormal?

Thirty-four years have passed since Wittmaack published his extensive studies on what he termed the normal and abnormal pneumatization of the temporal bone. His thesis generally has had a wide acceptance but has been modified since in some of its aspects by himself and by other investigators.

Fig. 1.—Embryonic myxomatous tissue lining the peri-antral cell wall of a child with acute otitis. X100. This type of tissue more or less fills the middle ear and antrum at birth. In the normal progress of pneumatization this lining contracts to develop into the thin mucoperiosteal lining as seen in Figure 2.

Previous to the publication of Wittmaack's work, the variations in the pneumatic structure of the temporal bone were apparently not in any way regarded as the result of disease. Wittmaack contended that any mastoid structure that did not develop a fully pneumatic pattern by the end of the fifth year of life represented a case of arrested pneumatization resulting from a pathologic process. He found the cause in a neo-natal otitis media resulting usually from the irritating effects of the solid constituents of amniotic fluid, from meconium, vernix, etc. aspirated through the eustachian tube, or from the occurrence of an otitis media during the nursing life of the infant. The influence of these events on the epithelium and subepithelial connective tissue of the preformed cavities, the middle ear and antrum (Fig. 1), and the role of this epithelium and its submucosa in the normal process of pneumatization determined whether the pneumatic process would be complete with a thin lining of mucoperiosteum in the middle ear, antrum and the cells (Fig. 2), or whether diploic structure would persist in a variable amount in the space normally occupied by pneumatic cell structure, and the lining

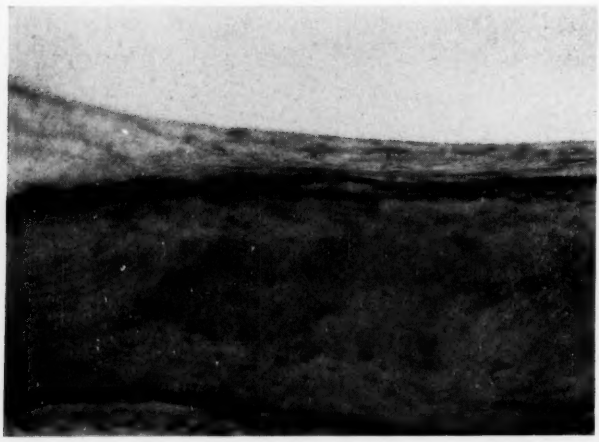


Fig. 2.—The epithelium is quite flat with long nuclei remindful of endothelium. The basement membrane lies directly on the delicate periosteum which has only one layer of cells similar to those at the surface. A longitudinal section of a vessel is seen in the submucosa. X200. When inflammation or constitutional factors affect the progress of the development of this thin mucoperiosteal lining, a hyperplastic fibrous mucous membrane may result as shown in Figure 3.

of the middle ear, antrum and what cell structure was present would have an abnormal amount of fibrous tissue in it, or appear predominantly hyperplastic and covered by a layer of cuboidal shaped epithelial cells (Fig. 3).

Other investigators, notably Albrecht and Schwartz, have called attention to hereditary and constitutional influences on the process of pneumatization. Albrecht and Schwartz together made an extensive study of the histological structure of the middle ear mucous membrane in 145 specimens of new born infants, and in the foetus. They found great individual differences in the activity of the epithelial cells and of the underlying layer of fibrous tissue, and in their opinion these differences could only be explained as inborn variations characteristic for each individual.

In a recent study entitled, "The Influence of Heredity on the Pneumatization of the Temporal Bone," H. W. Schwartz of Halifax, in his presidential address before the Canadian Otolaryngological

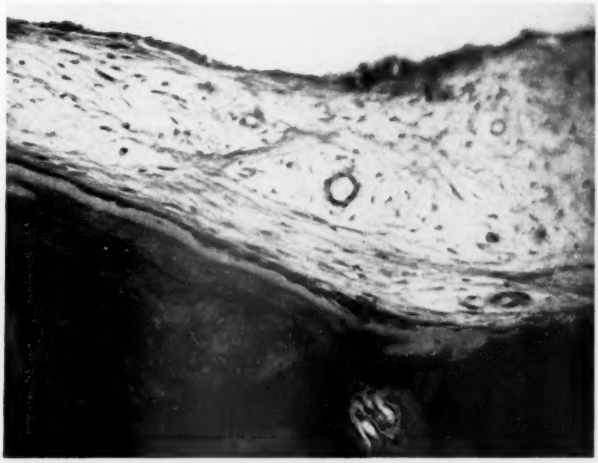


Fig. 3.—The epithelium is of the low cuboidal type. Between the epithelium and sharply defined periosteum, the connective tissue is poorly vascularized. It is of a clear cut fibrillary character, formed by long drawn out spindle cells. This mucous membrane is increased in thickness without edema by the formation of solid fibrous tissue. There is a patch of cylindrical ciliated epithelium visible. X200.

(These illustrations are from the collection of Doctor George Kelemen to whom the authors are indebted for their use.)

Society, summarized some evidence to show that a purulent otitis media in infancy may have no retarding effect on the process of pneumatization, and that acute inflammatory disease usually leaves little or no evidence recognizable in x-ray films taken six months to twelve months later. Schwartz studied the mastoid x-rays in sets of identical twins and in family groups and concluded that the high proportion of similar patterns in the mastoid processes of identical twins is strong evidence of heredity being a and probably the deciding factor, and that the classification of temporal bones of family groups, as determined by x-ray, also leaves the impression that heredity is probably the explanation of the different types of bone and the extent of pneumatization when present.

Probably then, some would consider it incorrect to refer to limited pneumatization of the temporal bone as strictly abnormal

in the instances where hereditary factors may have played the deciding role. The fact seems to have been established, however, by Wittmaack that it is the very thin mucosal lining consisting of a scantily vascularized fibrous layer covered by simple squamous epithelium that is normal, and that it is only in the well pneumatized mastoid that this mucoperiosteum predominates.

One can then argue that if in the lining of the middle ear and antrum of those temporal bones with limited or absent pneumatization there is a considerable amount of hyperplastic tissue, pure fibrosis, or even the persistence of some embryonic myxomatous tissue, as has been reported, this then is part of an abnormality regardless of whether this condition is the result of acquired or hereditary factors. The ophthalmologists have standards of normal vision. A certain percentage of the population they deal with have myopia. That is, I believe, considered to be an abnormality. Factors of heredity play a role in the incidence of this disorder. Cannot that analogy be applied to the incidences of limited to abnormal pneumatization in those situations where heredity may have had a prominent part?

How common is abnormal pneumatization in children growing up in this era of antibiotics? Our curiosity in this led us to make a small study, the results of which were as follows:

One hundred children over the age of five found in the wards of a public hospital and in whom there was no ear disease were subjected to mastoid x-rays in the Law position. Fifteen per cent showed very limited to absent pneumatization. In 10 of the 15 this was bilateral.

The x-rays of 100 consecutive children of a similar age group (past their fifth birthdays) who had been examined for acute head injury in a childrens' hospital were studied. The various positions taken allowed adequate visualization of the mastoid pneumatization. Thirteen per cent showed very limited to absent pneumatization. In 8 of the 13 this was bilateral.

One hundred students of college age, without selection, most of them under twenty, were subjected to mastoid x-rays in the Law position while undergoing their general physical examination. Fourteen per cent showed very limited to absent pneumatization. In 6 of the 14 this was bilateral.

This study would indicate one simple fact, that in a population between the ages of 5 and early adult life approximately 14 per cent probably have a middle ear mucosa in one or both ears that is not the thin mucoperiosteum of the well pneumatized mastoid.

Now let us examine this idea current among some otologists that the present day treatment of otitis media results in some instances of middle ear morbidity characterized by residual sterile secretion, and its sequelae. Some believe that it may even excite in time a pathogenic process leading to cholesteatosis.

Reference is common to "inadequate antibiotic therapy." By that is meant, supposedly, the limited use of an antibiotic for two or three days during the acute manifestations of an otitis media. The treatment is often prescribed over the telephone by the family doctor or pediatrician. The patient in these instances often does not have the benefit of an ear examination and rarely a follow-up test of his hearing ability. These questions are suggested:

"Would prolonged antibiotic therapy for a week or more insure complete resolution?"

"Should more attention be given to the possibility that the organism causing the infection may be resistant to the antibiotic being used?"

To us it has seemed that these are important considerations in certain individual cases; probably in all cases it is important that the antibiotic should be continued in an adequate amount longer than for one or two heavy doses of penicillin or for a day or two of one of the orally administered antibiotics currently popular. But we have decided that it is of equal importance to consider the matter of middle ear drainage. With early myringotomy, many of these cases resolve promptly with a minimum of antibiotic therapy. Without insured drainage, a certain percentage show up in the otologist's office, a month or two after the initial treatment with evidence of an exudate in the middle ear and a hearing level approximating the 30 decibel line through the speech frequencies. We then carry out myringotomy together with aspiration of middle ear fluid. Resolution seems to be prompt in cases in which the mastoid is well pneumatized. We have observed that it is uncertain when abnormal pneumatization is present.

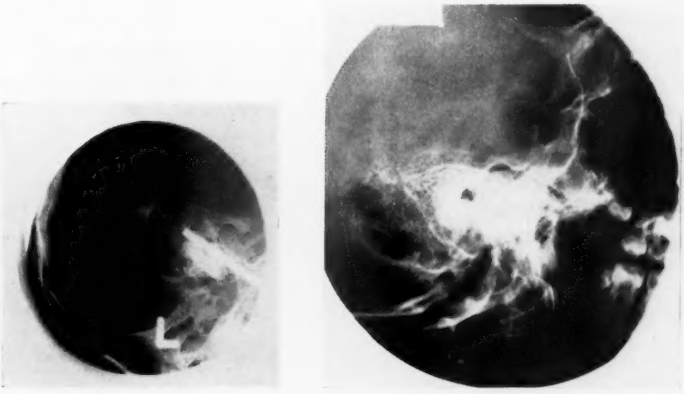
Reference was made earlier in this discussion to the matter of simple effusion of serum into the middle ear following eustachian tube obstruction. We have observed that the resolution of this condition is not a common problem in well pneumatized temporal bones. In fact it is less likely to occur and to persist when there is extensive pneumatization. This may be partly on the basis of pure mechanics in the production of the negative pressure considered to be a factor in the production of a transudate. We have also observed that in cases where the usual therapy such as inflation, myringotomy once or many times, tonsil and adenoid surgery where indicated, other attempts to control lymphoid hyperplasia such as with irradiation, and allergic management have failed, one will invariably find poor pneumatization when the mastoids are x-rayed.

Our contentions indicate that we believe that abnormal pneumatization is an important factor in the chronicity of certain cases of sterile fluid in the middle ear, and that it may play an important role in those children who develop chronic suppuration in this era of antibiotics. These conclusions are based on clinical observations.

The writings of Tumarkin have been focusing the attention of otologists on the problem of present day middle ear disorders. He subscribes fully to Wittmaack's views, recognized the role of abnormal pneumatization, claims that modern antibiotic therapy in its present form is one etiologic factor in attic suppuration and directs attention to the epitympanic space where he believes cholesteatosis may begin through metaplastic changes in the pavement epithelium of this area. He reports success with surgical therapy directed at the epitympanic space in cases of persistent fluid in the middle ear in the form of effusion that has resisted the usual method of therapy, as well as in treatment of attic suppuration with cholesteatosis.

The epitympanic space may well be the objective of the middle ear activity of otologists in the near future. That idea suggests the importance of the management of middle ear problems by the physician trained in otology, and a reconsideration of the role of abnormal pneumatization in these problems.

A possible advance in x-ray visualization of the pneumatic development of the mastoid may be an aid to the detection of minute pathologic changes in the mastoid. A new technique has been devised which "blows up" the detail two and one-half times. A special



A B

Fig. 4.—Roentgenogram in the Law position with the usual technique A, and the new technique B.

tube with a focal spot of 0.3 mm as contrasted with the usual focal spot of 1.0 mm is used and the exposure is made with the film 30 inches away from the skull instead of in contact. The "blown up" minute details are very prominent if the technique is correct. An enlargement of the usual film would not be comparable because this would enlarge the grain of the film and produce artefacts from dust and dirt. The following illustrations show views of the temporal bone in the Law position with the usual technique (Fig. 4A) and with this new "blown up" technique (Fig. 4B).

SUMMARY

There is evidence that limited or absent pneumatization of the temporal bone may be the result of one or more of the following factors:

1. Neonatal incidents causing irritation and inflammation of the lining of the middle ear and antrum.

2. Middle ear infection in early infancy.

3. Hereditary or constitutional factors. Probably this factor is an important one.

There seems to be agreement that when limited or absent pneumatization exists that the mucoperiosteum of the middle ear and antrum and what cellular structure is present is predominantly of a hyperplastic or fibrous type and is not the thin mucoperiosteum found in well pneumatized bone. In that sense the use of the term abnormal pneumatization is probably correct.

Observations have been made to indicate that this limited or absent pneumatization of the temporal bone will be found to be an important factor in:

1. Chronic cases of secretory effusion or serious otitis media, the cure for which is not found in the usual investigation and therapy.

2. The development of chronic suppuration of the middle ear in this day.

There seems to be evidence that the therapy now commonly used in acute otitis media without regard to drainage is followed in some instances with permanent changes in the middle ear and mastoid mucosa. This may produce a degree of permanent hearing loss. It also may produce a degree of vulnerability to subsequent infection in this mucosa.

If progress is to be made in preventing hearing loss from middle ear disorders and in preventing the evolution of chronic middle ear infection, the management of these disorders will need the critical evaluation of the physician who has a special knowledge of otology.

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DISCUSSIONS

DR. GEORGE E. TREMBLE: I heartily endorse what Dr. Boies has said here today. The actual cause of limited or absent pneumatization of the temporal bone is still unanswered and, like Dr. Boies, I feel that it most likely is the result of one or more of the factors which he has discussed. I do not think that we can definitely say that irritation, infection or heredity separately are dominant factors, but the most probable cause is a varying combination of these three conditions.

I was interested in hearing the speaker use the term cholesteatosis in place of cholesteatoma. It seems a better word. The ending—oma comes from a Greek word which suggests "a swelling" and this has been associated in medical terminology with tumor—such as lipoma or granuloma. There are exceptions of

course such as glaucoma and trachoma but in the main the soft mass which invades and hollows out the hard dense mastoid bone resembles a growth.

The modern view, however, is that cholesteatoma is not a tumor and that the ending—osis which means "containing" or "full of" is more descriptive.

The term cholesteatosis seems more in accordance with recent theories of the pathogenesis of this condition.

I noted his reference to Tumarkin—the latter feels that the most important cause of abnormal pneumatization is latent mastoiditis associated with gastroenteritis in nursing infants. In this condition, Tumarkin thinks the mucous membrane of the tympanic cleft becomes in some way devitalized and so, in later life, is predisposed to breakdown under the impact of infection. He has shown that in the middle ear there are two types of mucosa—the tympanic mucous membrane, covered by a ciliated lining and the epitympanic area or attic, covered by squamous epithelium and that in adverse circumstances these two types behave in different ways. The ciliated epithelium around the opening of the eustachian tube gives rise to a mucopurulent type of otitis media while the squamous epithelium tends to proliferate and form heaped up layers of pavement cells. These squames, as he calls them, according to Tumarkin, are responsible for all the various pathological conditions found in the chronically infected mastoid.

Wittmaack holds that due to the insults of infection new connective tissue is formed beneath the epithelial layer of the advancing antral sac and as a sequelae of this, by a mechanical process, further pneumatization is prevented.

Albrecht feels that heredity is the most important factor and during the past 10 years Dohlman has stressed the role of allergy—which can be considered under "heredity."

The question of fluid in the middle ear is very important. As early as 1683 Duverney recorded the presence of fluid in the middle ear of the new born. Since that date liquor amnii vernix caseosa, meconium, milk and even pus have been reported in the tympanic cavity.

In looking over my notes of Professor Ruttin of Vienna, he stated that histologically 60% of all infants at birth show foreign body otitis media but clinically these otites are usually overlooked. The importance of such cannot be denied but it is probably not the primary cause.

Again as Dr. Boies has mentioned, I would like to stress that the etiology is likely multiple and not due to any single factor.

I would like to take this opportunity to thank the speaker for allowing me to discuss his paper.

DR. KENNETH M. DAY: I have been interested in this pneumatization problem for many years and discussed it some ten years ago in a paper on primary pseudocholesteatomas before the Triological Society. I still believe that normal pneumatization of the temporal bone is dependent upon proper ventilation of the middle ears in infancy. Routine examination of the ears of infants and young children reveals a surprisingly large number with nonventilated or secretory middle ears. In my experience fully 80% to 85% of the cases of secretory otitis have an allergic basis. The hereditary tendency to have imperfectly pneumatized temporal bones may well be due to the hereditary tendency to allergic reaction.

As to perforations of Shrapnell's membrane and the development of cholesteatomas I wonder how many of you have ever seen such perforations occur. They

never occur with acute middle ear infections. I have seen perforations of Shrapnell's membrane occur in five cases of secretory otitis media with previously intact drums. In two of these cases the perforation later closed, one of them spontaneously and the other following removal of a small polyp. It seems to me that it would be quite possible for squamous epithelium to invade the attic in either of these cases and later develop into a cholesteatoma behind an intact drum and Shrapnell's membrane.

I believe that the pouching of Shrapnell's membrane with or without rupture is due to lack of ventilation of an attic which is sealed off from the middle ear below as a result of secretory otitis. In such a case the mastoid will be nonpneumatized or poorly pneumatized depending on the age at which the secretory otitis occurred.

It seems quite unnecessary to theorize about metaplasia of the attic mucosa as propounded by Tumarkin and others in order to explain the development of attic cholesteatomas.

DR. PHILIP E. MELTZER: You heard this morning that there were some 12 theories of hearing and a 13th in the process of publication. I know of five theories of pneumatization, and probably there are more that I don't know about. Why assume that the Wittmaack theory is right and the others wrong? It would appear from the paper of Dr. Boies and the discussion by Dr. Tremble that they are adherents of the Wittmaackian theory. There are many who are not adherents of that theory. There are many who believe that the variations in the mucosa of the middle ear are constitutional and they have just as much reasoning behind their belief as has Wittmaack. Cheatle, who proposed that the compact mastoid was normal for that individual and variations in pneumatization were normal variations, has many adherents of his theory. Throughout the paper I gathered any pneumatization differing from the truly pneumatic was abnormal and that the mucosa of the middle ear and antrum was therefore abnormal. I is quite possible that this type of mucosa may, when moderately involved from whatever the cause, result in accumulation of fluid in the middle ear and may require the kind of treatment that Dr. Boies outlined. I would not personally assume that such fluid accumulation is due to abnormal mucosa and pneumatization.

DR. LAWRENCE R. BOIES: I do not accept the Wittmaack theory as a complete explanation of variations in pneumatization. I believe that heredity plays an important part in the incidence of these variations. Apparently I did not make this point clear. However, on the basis of the evidence accumulated to date, it would seem that when there is very limited or absent pneumatization the lining of the air containing cavities is not the thin mucoperiosteum found lining the cells of a well pneumatized bone, but tends to be a fibrotic or hyperplastic type. My thesis is that when infection occurs in that type of soil, or a transudate results from Eustachian tube blockage there is a greater tendency for the condition to become chronic. This to me is a partial explanation of certain of our chronic problems.

LXXIII

THE PROBLEM OF SYNKINESIS AND CONTRACTURE IN CASES OF HEMIFACIAL SPASM AND BELL'S PALSY

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There are two types of spasm affecting the face which are of special interest to the otologist because they seem to be the result of injury to or irritation of the facial nerve in its passage through the temporal bone. This part of the facial nerve usually is divided into the portions in the internal auditory meatus, the porus acusticus internus, the geniculate ganglion portion, the tympanic or horizontal portion, the vertical portion and the pes anserinus.[1]

The two types of facial spasm of interest to the otologist are those classified by Wilson and Bruce[2] as cryptogenic and symptomatic. Features of the typical one-sided form of cryptogenic spasm comprise tonicoclonic contractions of muscles from frontalis to platysma, notably those at the eye and mouth. Irregularity in site, rhythm and degree is the salient feature. The movements are evoked or intensified by voluntary acts, such as speaking or eating, or by involuntary acts, such as laughing; rest exerts a calming influence but psychic strain does the reverse, as when, for instance, the patient runs the gauntlet of inquisitive eyes in a public conveyance. In sleep, as a rule, the spasm dies down. Under the classification of "cryptogenic" or "primary facial spasm" Wilson and Bruce also included blepharospasm. This, they felt, was caused by a branch involvement of the lower motor neuron and tended gradually to involve branches of the facial nerve to the rest of the facial musculature.

Under the title of "symptomatic facial spasm" Wilson and Bruce described postparalytic facial spasm. They stated that this

type, known since the days of Charles Bell and Marshall Hall, now and again follows facial paralysis at a somewhat lengthy interval, and assumes a variety of forms. Mostly combined with contracture, although at times occurring without it, this type is seldom so severe or so widespread as the cryptogenic. The authors wrote that the postparalytic form can be distinguished from the primary cryptogenic form chiefly by the history but also by the constant accompaniments of muscular weakness, overaction and (generally) contracture.

In reviewing 106 cases of primary or cryptogenic facial spasm, Ehni and Woltman[11] said that they chose the term "hemifacial spasm" to describe the disorder because the condition is one that characteristically affects half the face.

They gave the following characteristics as being of importance in recognizing primary or cryptogenic hemifacial spasm: (1) the spasms are of an intermittent, twitching nature; (2) the eyelids on the homolateral side are almost always involved; (3) the spasms usually are unilateral and when bilateral they are not synchronous or equal in extent or severity; (4) the spasms may persist in sleep; (5) the patient does not feel any compulsion to make the movement; (6) the patient is unable to stop the movement by exercise of the will; (7) the patient cannot reproduce the movement voluntarily; (8) psychic upsets of any sort, fatigue and voluntary movements of the face, make the condition worse; (9) children do not have hemifacial spasm and (10) the spasms are limited to muscles innervated by the facial nerve.

The available evidence suggests that the hemifacial spasm which follows Bell's palsy and the hemifacial spasm called "cryptogenic" are clinically indistinguishable except by history, because muscular weakness and contracture are equally prevalent in both. The evidence also indicates that the etiology of each condition may be the same.

Two hypotheses have been offered as to the cause of hemifacial spasm of both the primary or cryptogenic type and the postparalytic type. The first offered suggests that hemifacial spasm is a central effect due either to degeneration of cells in the rolandic motor cortex or to degeneration of cells of the facial nucleus in the medulla. The second hypothesis offered is that hemifacial spasm is a disorder of the lower motor neuron.

Ballance[4] and Sullivan[5] both suggested that these phenomena are really local epileptic phenomena which are caused by disturbance of cell function. Ballance[4] submitted sections which were said to demonstrate that there were regressive changes in the pyramid cells of the facial area of the opposite rolandic cortex and that there were also regressive changes in the cells of the facial nucleus in the medulla.

Sullivan[5] embraced this concept of Ballance, but stated that regressive changes in the facial nucleus in the medulla are the essential cause of the muscular spasm of the face. He felt that they never are the result of obstruction to the passage of nerve impulses traveling along nerve fibers, but rather, a disorder of the cells of the facial nucleus surrounded by tissue and vascular changes. He suggested that a period of six months be allowed to elapse before decompression of the facial nerve in patients who have partially or completely unresolved Bell's palsy, for such a period would allow the facial medullary nucleus to atrophy and thus prevent the development of facial tic. Ballance,[4] on the other hand, wrote that the facial tics in question develop in those cases of Bell's palsy in which operative interference has been too long delayed. He appeared to feel that early operation would tend to prevent the development of tic by earlier restoration of function to the nuclei.

In discussing Sullivan's paper, Tickle[6] remarked that a tic had developed in a large number of his patients who had been operated on for relief of Bell's palsy of more than six months' duration.

In the discussion of the same paper, Fowler[7] declared that he had been unable to substantiate the findings of Ballance in regard to regressive changes in the facial area of the rolandic cortex after section of the facial nerve. He stated that Ballance had sent him the sections on which he based his hypothesis. All the pyknotic cells which Ballance showed on the "degenerated" side were said to be on the edges of the section, and the normal ones he showed on the intact side were said to have been taken from the center of the section. Fowler noted that it is a very common thing for the edges of a piece of brain tissue to exhibit pyknotic changes.

The weight of the evidence seems to be in favor of the hypothesis that hemifacial spasm is a lesion of the lower motor neuron. For instance, Ehni and one of us (Woltman[3]) felt that their case of hemifacial spasm in which hemiplegia presumably due to capsular infarction failed to affect the spasm, and a similar case of Habels,

constitute evidence that the lesion is not situated in the corticobulbar pathway. A number of circumstances, they felt, point to the final common path to the facial muscles as the site of the lesion. In hemifacial spasm the frequent occurrence of weakness and contracture of muscles supplied by the facial nerve speaks strongly for disease of the lower motor neuron. Also, the undeniable fact that gross lesions of the lower motor neuron produce twitching of the face very much like, if not indistinguishable from, the twitching of cryptogenic spasm, makes it seem highly probable that the lesion causing hemifacial spasm lies somewhere between the facial nucleus and the stylomastoid foramen. In discusing muscle spasm occurring during recovery from facial palsy, Duel[8] stated that in all the monkeys in which a graft of the facial nerve had been placed, an associated muscle spasm or tic developed gradually, becoming more exaggerated as facial movements increased up to a certain state, and then gradually decreasing as facial control became more and more normal. Yet in all cases the facial spasm remained in some degree. He said that the type of muscle spasm seemed analogous to that frequently observed in partially abated Bell's palsy in human beings.

In a series of experiments designed to test the validity of these hypotheses of Duel[8] and Sullivan,[5] Fowler[9] and Howe, Tower and Duel[10] seem to have made the hypothesis of the central origin of these spasms completely untenable.

In a paper on abnormal movements following injury to the facial nerve, Fowler[9] stated that monkeys on which a graft of the facial nerve had been done were allowed to recover after removal of the facial area in the rolandic cortex. The ticlike movements of the face were found to persist without change. To make sure that the result was not the result of incomplete ablation of the rolandic cortex, in 2 monkeys the entire rolandic motor area was removed, and in 4 others the entire cerebral hemisphere was removed. The ticlike movements of the face again were found to persist without change. In 2 monkeys, when the cerebral cortex was removed from both sides, it was difficult to elicit the facial tic, but associated movements were persistent on the side on which the facial nerve had been cut and repaired. Fowler and Wolfe again were not able to agree with Ballance[4] as to the changes observed by him in staining sections of the motor rolandic cortex. They said that the Nissl reaction is notoriously difficult to interpret, and in their opinion the changes reported by Ballance are simply normal variations due to microscopic

technic and inherent in the method, particularly when small specimens are used.

Howe, Tower and Duel[10] reported the findings of an experimental study on facial tic in relation to injury of the facial nerve in 27 rhesus monkeys. In these monkeys the facial nerve was injured just distal to the stylomastoid foramen by (1) complete section and resuture, with or without the insertion of a nerve graft; (2) partial section; (3) freezing or (4) injection of alcohol. Soon after the first evidence of returning function, they observed purposeless twitching movements which involved various muscle groups and at times the entire area, which previously had been paralyzed. They found that in general the gravity of the tic was proportional to the degree of disturbance in the bed of the facial nerve, rather than to the amount of the paralysis.

Howe, Tower and Duel[10] did four further series of experiments. First, in 4 animals the electrically excitable cortex of the frontal and parietal lobes was stimulated with a 60-cycle current of from 0.5 to 2 milliamperes. Many normal facial movements resulted, but there was no suggestion of the tic, with the exception that stimulation of any point which caused closure of the lids brought forth an adventitious movement. Although the tic could be elicited by stimulation of the cortex, it persisted in the absence of the cerebral hemisphere.

Second, when the facial nerve was cut at the stylo-mastoid foramen or at its point of exit from the medulla, stimulation of the central end of a cut branch produced contraction of muscles supplied by other intact branches. The mechanism of adventitial facial movements was thereby shown to operate entirely in the periphery. The authors wrote that in this instance the presence of branched axons offered the only hypothesis that could account for the phenomenon on an anatomic basis. On the normal side, however, stimulation of a branch nerve never gave a mass contraction of the entire facial field, as it did on the side of operation.

It remained to demonstrate branching axons histologically. A specimen of the injured nerve containing the site of injury was removed. After staining by the silver impregnation method, bifurcating fibers were commonly found, and numbers of axons were seen arborizing into three to four branches.

Howe, Tower and Duel[10] cited the work of Ramón y Cajal[11] as demonstrating the fact of branching of axons at the site of injury of a peripheral nerve. In reviewing the work of Ramón y Cajal,[11] however, it should be noted that although he described the branching of axons at the site of injury, he did not trace the branched axons to their peripheral distribution. Howe, Tower and Duel[10] did not demonstrate anatomically that branching axons reached a different peripheral distribution than did the original intact axons. They assumed that their physiologic experiments had demonstrated this.

Howe, Tower and Duel[10] concluded, as a result of the foregoing experiments, that single regenerated axons had by branching come to supply widely separated muscles, and that an irrevocable functional union had taken place between muscles which do not normally contract simultaneously. Fowler,[9] as well as these authors, appears to feel that there is no alternative hypothesis which could explain the experimental findings.

In investigating the effect of injury on mammalian nerve fibers, Adrian[12] found that such nerves, set up in a warm, moist atmosphere, usually give rise to persistent rapid fluctuations of potential which are caused by a persistent discharge of impulses in some of the fibers. The persistent discharges arise from the injured ends of nerve fibers, the permanent depolorization of the injured region acting as a stimulus to the intact part of the fiber. Adrian found that one factor affecting the results of injury may be the presence of dense layers of tissue near the injured region of the fibers. The difference between large and small nerves can be shown in the case of motor discharges. When the sciatic or main peroneal nerve is cut through in a spinal cat, the leg muscles give a contraction which does not subside for several seconds. But, as a rule, cutting of the small branches which supply each muscle gives no more than a brief twitch. This difference in reaction seems to depend mainly on the thick sheath of the larger trunks, for if this sheath is stripped away for a short distance in the middle of the nerve a cut above or below the stripped region will give only one or two responses. Stripping the sheath has the same effect on the persistent discharges recorded from the excised nerve, for these are abolished or greatly reduced by removal of the sheath for a centimenter or so from either end.

Granit, Leksell and Skoglund[13] discussed Adrian's findings and those of Hering, who cut the sciatic plexus of a frog, tied a knot

around the nerve at the knee and applied stimulation with a pair of electrodes just above the knot. Muscles above the knee were then found to contract, signifying that impulses caused by the stimuli had ascended the nerve fibers to the cut end of the plexus in the pelvis, there to pass over into adjacent motor fibers innervating muscles above the region of the knee. Nerve fiber interaction thus may take place at an injured region, which in this respect acts as an artificial synapse.

Granit, Leksell and Skoglund[13] commented that since the possibility of fiber interaction in a pathologically altered region of a nerve is a question of considerable importance in clinical neurology, it is surprising how little attention has been devoted to experimentation in this field and how commonly either central effects or fiber dichotomy are invoked to explain every apparent exception to the classical law of isolated conduction in nerve fibers. They found in experiments on cats that nerve impulses set up in a motor root are transmitted to the sensory fibers in a cut region of the nerve and can be picked up in the sensory root of the same segment. The cut region thus serves as an artificial synapse. Fiber interaction of this type, they found, also takes place at a crushed region, and, moreover, they found it present in a region subject to mechanical pressure of a moderate order. They concluded that an injured or compressed region forms a critical point at which impulses can be reflected centripetally or centrifugally by engaging other fibers by interaction.

In investigating injury activity and trigger zones in human nerves, Kugelberg[14] found that various symptoms of local irritation in nerves can be produced by compression ischemia. He was able to define some of the conditions under which an active focus in the nerve gives rise to spontaneous discharges, and described two different forms of spontaneous activity from the local focus caused by local ischemia. One form occurring during ischemia consists of isolated action potentials of irregular frequency. The second form seen after ischemia consists of repetitive discharges occurring in bursts.

Although it seems evident that Howe, Tower and Duell[10] have excluded the possibility that the central effects postulated by Ballance[4] and by Sullivan[5] can be present or can account for the spasm or the synkinesis, their work does not exclude the possibility that repeated discharges arising from the injured area of the nerve may

be the cause of the spasm or that fiber interaction occurring at the injured area may account for the synkinesis.

Spiller[15] suggested that the cause of contracture occurring in partial recovery from paralysis of the facial nerve is the result of overstimulation of the affected muscles. The fact that a state of continuous contraction is present, he said, means that complete relaxation either is not obtained or is obtained for a much shorter period than for normal persons. The affected muscles, kept in a more contracted state, gradually shorten, and in this way the late contracture is produced. Assuming that Spiller's hypothesis is correct, the repeated stimuli arising from an injured region of the facial nerve would be a sufficient cause for such contracture.

While a hypothesis of ischemic injury to the facial nerve would serve to explain the spasm, synkinesis and contractures seen in hemifacial spasm and Bell's palsy, it remains to be suggested how such ischemic injury might arise.

Audibert, Mattei and Paganelli[16] wrote that the only reasonable explanation of the symptoms of Bell's palsy is dysfunction of the autonomic system, a dysfunction which produces spasm, thrombosis, or embolism in the blood supply to the nerve. They felt that the facial nerve is particularly susceptible to vasomotor disturbances because its blood supply comes in part from the middle meningeal artery and in part from the stylomastoid artery. They thought that if the middle meningeal artery is involved, to the motor paralysis are added changes in hearing, in taste and in salivary secretion. If, on the contrary, the stylomastoid artery alone is involved, there would result only a pure motor paralysis. They also considered the possibility that both the stylomastoid and the middle meningeal supply might be involved at once. They felt that if cold is a factor in precipitating the disorder, it acts only by producing an autonomic disequilibrium, a physical allergy to cold.

For treatment they advised the use of vasodilators, purgation and a diet low in sodium. Müller[17] found that vasomotor disturbances are particularly likely to occur at the junction of arterial supplies.

Kettel[18] accepted the hypothesis of Audibert, Mattei and Paganelli,[16] and supported their hypothesis with the findings at operation on 50 patients who had Bell's palsy. In all the patients he found granular decay of bone and necrosis of bone, but no cellular infiltration.

He concluded that the primary and central feature of Bell's palsy is a disregulation of the circulation which probably takes place near the stylomastoid foramen. The result is ischemic paralysis and ischemic bony necrosis. He felt that possibly there is a cold allergy related to that of Ménière's disease.

In considering the hypothesis that vasospasm may be the fundamental cause of Bell's palsy, it may be pointed out that vasospasm sufficient to produce necrosis, when it has been observed elsewhere in the body, appears to be associated with tissue injury of a sort which maintains a reflex to the affected vessel.

It might be considered reasonable to suppose, therefore, that in Bell's palsy and primary or cryptogenic hemifacial spasm, even though vasospasm might play a part in producing the lesion, there might be a still more fundamental tissue lesion tending to set up a reflex, producing the vasospasm.

On this basis it occurred to one of us (Woltman) that the fundamental lesion in both hemifacial spasm and Bell's palsy might be a fibrous constriction of the sheath of the facial nerve somewhere in its course through the temporal bone, but probably in close relationship to the stylomastoid foramen.

OPERATIVE FINDINGS IN HEMIFACIAL SPASM

The feasibility of decompressing the facial nerve in its canal was discussed with and accepted by 7 patients with hemifacial spasm and 1 patient with blepharospasm. There were 2 men and 6 women in this series of patients. The hemifacial spasm was on the left in 4 instances and on the right in 4 instances.

Surgical Technic.—The first operation was done by means of an endaural incision; subsequent operations were done by the postauricular approach. The antrum of the mastoid was entered and the mastoid was exenterated until the bulge into the mastoid tip of the digastric fossa was located. The tip was removed up to this line, and the pes anserinus was identified as it entered the stylomastoid foramen. The bone of the posterior wall of the external auditory canal was then removed from the anterior surface of the vertical portion of the fallopian canal with a surgical engine and mill. Removal of bone began at the fossa includis and followed downward parallel to the vertical portion of the fallopian canal, an action accomplished by sweeping the mill (burr) around the curve of the pos-

terior bony wall of the external auditory canal. When sufficient bone had been removed to define the fallopian canal from the pes anserinus to the lower turn of the fallopian canal at the level of the horizontal semicircular canal, bone was removed from each side of the fallopian canal with a 3-mm mill. A thin dome of bone was left over the nerve. With a fine curet this bone was removed from the nerve; a start was made at the stylomastoid foramen and the work was carried upward. In the first operation the bone was removed from the anterior surface of the nerve at the lower turn and was carried forward to the region of the short process of the incus. Then the nerve sheath was grasped at the pes anserinus and the sheath was slit from below upward with a myringotomy knife. The edges of the slit sheath were then turned forward and backward, so that the nerve fibers were exposed.

Because at the first operation pathologic changes in the nerve appeared to be confined to the region of the stylomastoid foramen and the first centimeter or centimeter and a half immediately above it, all subsequent surgical approaches were made through a postauricular incision, since it was felt that operation on the lower portion of the nerve could be more rapidly done through this approach. In 1 case the nerve sheath was ballooned up by an injection of isotonic solution of sodium chloride into the nerve sheath before it was slit.

FINDINGS AT OPERATION IN REGARD TO APPEARANCE OF NERVE

CASE 1.—At the stylomastoid foramen there was found attached to the inner sheath a fibrous nodule about 2 mm across.

CASE 2.—The facial nerve did not appear to be especially abnormal, except for the fact that the sheath was somewhat tough and resistant to slitting.

CASE 3.—The fibrous envelope at the stylomastoid foramen was very tough for a distance of half a centimeter, resisting division. Above this area no sense of resistance was felt.

CASE 4.—The sheath and dilated portion at the pes anserinus appeared neither swollen, reddened nor with any sort of an abnormal appearance.

CASE 5.—At the pes anserinus the sheath of the nerve was thick, and definitely resisted the knife. A little further up the sheath became very thin, and could be separated without resistance.

CASE 6.—The entire sheath of the nerve felt unusually thick and resisted the knife.

CASE 7.—At the stylomastoid foramen there was a very marked thickening of the sheath and it was very tough. After it had been slit for a centimeter, it became very thin.

CASE 8.—There was nothing suggesting an abnormal appearance. Before the sheath was slit, it was distended with saline solution. It was divided with ease.

PREOPERATIVE HISTORY AND FINDINGS

CASE 1 (*operation on the left*).—The onset of twitching about the left eye had been noted five years before admission of the patient. It had continued to progress slowly until the entire left side of the face was involved to grade +2 to +3.

CASE 2 (*operation on the right*).—A spasm had started about the right eye eight years previous to examination of the patient here, and had gradually spread to involve the entire face. There was no involvement of the ear or other structures.

CASE 3 (*operation on the right*).—This patient stated that the difficulty had started with twitching of the right eye six years before she came to the clinic. A year before the operation alcohol had been injected into the second division of the right facial nerve. This had produced grade −3 weakness of the right side of the face and relief of the spasm for three months. After the return of function, the spasm was more frequent and severe, and spread to the entire right side of the face, with contracture and associated movements.

CASE 4 (*operation on the left*).—The spasm had started around the left eye three years previously, and gradually had spread to the mouth. A year prior to the patient's admission to the clinic alcohol had been injected; it gave her relief for six months. She had a sense of clicking in the left ear during the spasm.

CASE 5 (*operation on the right*).—Twitching of the right side of the face had started four months before the patient's admission here. It had begun in the right eye and gradually had spread to the rest of the face. She had had twitching two years before; it had lasted three weeks. There were associated movements. When the brow was raised, the lips moved to the degree of grade +2; when the lips were pursed the eye closed to the degree of grade +1. A

month before admission the patient began to have volleys of flashing pain above the right eye, like the pain of trifacial neuralgia but without the trigger action. This had disappeared the next day, after two injections of a sedative agent.

CASE 6 (*operation on the left*).—The spasm had started in the left side of the face fifteen years previously. Three years intermittent pain had developed in the right upper trapezius region.

CASE 7 (*operation on the right*).—Hemifacial spasm had started seventeen years previously. There were considerable contracture and marked synkinesis with all movements.

CASE 8 (*operation on the left*).—The left side of the face had started to twitch three years prior to the patient's coming to the clinic, and gradually had become worse. The platysmal part of the spasm produced the most distress.

POSTOPERATIVE PROGRESS

CASE 1 (*operation on the left*).—Six days after operation there was a minimal residum of the spasm in the forehead. Voluntary power in the forehead was grade −3 to −4. Power elsewhere in the facial supply was grade −4. Three days later there was no twitching, and voluntary power over the facial supply was grade −2 to −3.

Two months later the voluntary power in the left side of the face had almost entirely returned, except for very slight weakness at the angle of the mouth.

Eleven months after the operation the patient reported that the twitching in the left eye had begun to return.

At nineteen months postoperatively the patient reported some twitching of the face for a few days at a time, when he had a stuffy feeling in his head with sneezing. The twitching would stop for perhaps a week, then start again and persist for a few days. When present, however, the spasm was only half as severe as before surgery. There had been no disturbance of taste, but at times there was some difficulty in hearing.

CASE 2 (*operation on the right*).—A little spasm was present at intervals for two days after the operation. On the seventh post-

operative day power to elevate the muscles over the right forehead was grade −1 to −2; closure of the eyelid, grade −3 in power and grade −2 in amplitude; showing of the teeth, grade −2 to −3 in both power and amplitude. There was no twitching and no associated movement. The patient reported loss of hearing on the right and dysgeusia.

Eleven months after the operation the patient wrote that her spasm "was picking up about where it left off at the time of operation." There was still some loss of hearing and tinnitus was present, as was dysgeusia. The return of the spasm would be preceded by occasional outpouring of saliva from the parotid gland. This does not occur with spasm.

Fifteen months after the operation the patient reported buzzing and loss of hearing in the ear on the side of operation and said that the lower right jaw and tip of the tongue seemed to ooze a queer-tasting, burning saliva. The spasm associated movement and contracture were as severe as, or possibly worse than, before the operation.

CASE 3 (*operation on the right*).—The day after operation spasm was not present, and there was only a grade −1 weakness of the entire face. Two days later the weakness was much more marked. Ten days after the operation twitching was not present. Weakness over the distribution of the right facial nerve was about grade −3 over the forehead, and grade −2 to −3 elsewhere. There were no associated movements. Two months later the patient reported that twitching was not present, her sense of taste was normal and her hearing was somewhat diminished.

Nine months postoperatively the patient reported occasional twitches in the eyelids on the right, accompanied by a sensation of fluttering in the right ear.

CASE 4 (*operation on the left*).—Only moderate facial weakness developed at 2:00 p. m., after the operation, performed at 8:00 a. m. The next day the face seemed a little better, but on the seventh postoperative day facial weakness was complete, with disturbance of taste and hearing; there was no spasm. A month after the operation there was still weakness of the upper and lower lips. The skin over the forehead could be moved normally. The spasm began to return nine months after operation. Muscular strength in the

left side of the face was grade −1 to −2 in the forehead, and grade −1 for puckering the lips. When the brow was raised, there was slight synkinesis involving the lips, unless a spasm supervened. When the lips were pursed, there was grade +2 to +3 associated movement in the forehead and eyes. Contracture was graded +2. Taste and hearing were still abnormal.

CASE 5 (*operation on the right*).—This patient had a grade 1 weakness in the distribution of the right facial nerve on the day after operation; this weakness progressed to complete palsy by the third postoperative day. Ten days postoperatively she had grade −1 to −2 movement of the forehead and eyes and grade −3 to −4 movement of the angle of the mouth and lips. Four months later there was no return of facial spasm. She had no difficulty with hearing or sense of taste. There was still some weakness of grade 2 to 3 at the angle of the mouth and grade −1 to −2 of the eyelids. The patient reported that the movements of her face were normal six months after the operation. At ten months after the operation she had no facial spasm or other symptoms.

CASE 6 (*operation on the left*).—Facial palsy of grade −4 developed at 5:00 p. m. on the day of operation, but there was no twitching. The patient returned four months later in a state of severe depression. There was no spasm, but grade −1 to −2 weakness of the face was present, and was especially marked at the left angle of the mouth. There was no disturbance of taste. The patient reported complete loss of hearing on the left. Results of a Doerfler-Stuart test suggested psychogenic deafness on the left. It was said that the patient had attempted suicide. On psychiatric examination she was found to have a lifelong pattern which predisposed her to depressive reactions.

CASE 7 (*operation on the right*).—Facial weakness on the day after operation was noted. There was still some twitching on the right side of the face. All twitching appeared to have disappeared by the third postoperative day. Two months postoperatively the patient reported that hearing and taste were normal and that the spasm was gone.

Nine months after the operation the patient reported that no difficulty with hearing or taste had been noted since the operation. He reported a slight return of the spasm at certain times for the previous three months, but it had not awakened him from sleep as

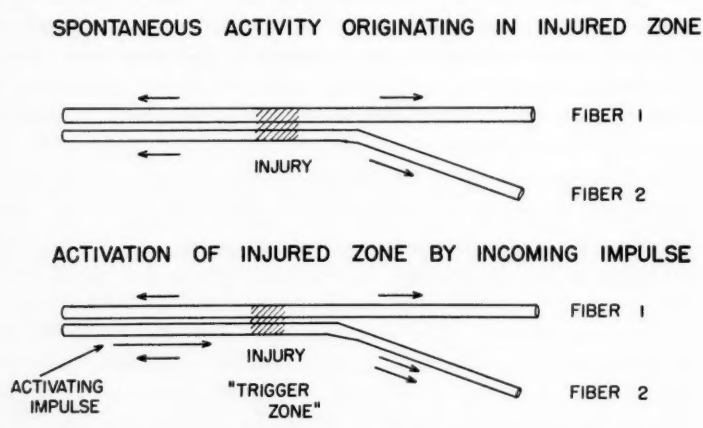

Fig. 1.—A possible mechanism of hemifacial spasm. Fibers 1 and 2 represent axons of the facial nerve innervating the upper and lower parts of the face, respectively. Local injury to the nerve by compression and ischemia in the facial canal is indicated by cross hatching. Upper diagram: Spontaneous discharge of impulses (the arrows) from the injured zone produces spasm or contracture. Lower diagram: During interparoxysmal periods, nerve fibers in the injured zone are hyperexcitable. The action current of a "voluntary" impulse in fiber 2 acts as a trigger to the injured zone, causing a discharge of impulses in both fiber 1 and fiber 2, whereas only the impulse in fiber 2 was willed. Synkinesis results from this interaction between nerve fibers. The effects of antidromic impulses in motor fibers and of impulses in sensory fibers have not been considered in this discussion. (Reproduced from: Woltman, H. W., Williams, H. L., and Lambert, E. H.: An Attempt to Relieve Hemifacial Spasm by Neurolysis of the Facial Nerves. Proc. Staff Meet., Mayo Clin. 26:236-240 (June 6) 1951.)

it did before the operation, and he estimated that he had obtained much relief.

CASE 8 (*operation on the left*).—The day after operation the patient had a grade −4 weakness of the entire left side of the face; there was no spasm. A year later the function in the face had not entirely returned, but she was still free of spasm.

COMMENT

The most interesting result of decompression of the facial nerve in the presence of hemifacial spasm was the disappearance of the synkinesis or associated movements of the facial muscles. Hereto-

fore, associated movements generally have been attributed to either branching or misdirection of nerve fibers during regeneration. In this case, however, different parts of the facial muscles, from frontalis to platysma, would become irrevocably linked to one another in their actions. Such an explanation of associated movements is not in harmony with the disappearance of associated movements after decompression of the nerve. Explanation on the basis of a reversible physiologic mechanism, rather than an irreversible anatomic one, must be sought.

A possible mechanism of hemifacial spasm which appears to fit the observations which have been made assumes the occurrence of spontaneous discharges of interaction of "cross firing" of nerve fibers in the facial canal. This is illustrated in Figure 1. Fibers 1 and 2 represent axons of the facial nerve innervating the upper and lower parts of the face, respectively. Local injury to the nerve by compression and ischemia in the facial canal near the stylomastoid foramen is indicated by the crosshatching.

In the upper diagram spontaneous discharge of impulses represented by the arrows occurs from the injured zone. It is this activity which may produce both the spontaneous spasms and the so-called contracture of the facial muscle.

In the lower diagram, during interparoxysmal periods when there is no spontaneous activity, the nerve fibers in the injured zone remain hyperexcitable. In this state, the action current of a normally initiated impulse in fiber 2 may act as a "trigger" to the injured zone. A discharge of impulses occurs in fiber 1 as well as in fiber 2, whereas only that in fiber 2 was intended. As a result of this interaction between nerve fibers, associated movements occur. Both upper and lower facial muscles contract, whereas only contraction of the lower muscles was intended. In this manner not only the spontaneous contractions, but also the associated movements in hemifacial spasm, may result from local injury to the facial nerve in its canal.

The occurrence of spontaneous discharges and fiber interaction in the presence of injured nerves certainly is not a new concept. These phenomena have been observed under experimental conditions since the very beginning of electrophysiology.[12, 19] Particularly in the past decade, however, experimental analysis has made it apparent that spontaneous discharge and fiber interaction may explain a num-

Fig. 2.—Patterns of discharge of motor units in the presence of hemifacial spasm. Records of the action potentials of motor units during spontaneous activity detected with a concentric needle electrode placed at the corner of the mouth. *a.* Irregular discharge at a slow rate; time signal, 1/10 second. *b.* Rapid regular discharge at 60 per second; time signal, 1/100 second. *c.* (*Both lower tracings*). Grouped discharges; the frequency of discharge in each group was up to 250 per second; repetition rate of groups was 1 to 5 per second; time signal, 1/1000 second. (Faint portions of lines were retouched in the photograph to enable tracings to be reproduced.) (Reproduced from: Woltman, H. W., Williams, H. L., and Lambert, E. H.: An Attempt to Relieve Hemifacial Spasm by Neurolysis of the Facial Nerves, Proc. Staff Meet., Mayo Clin. 26:236-240 (June 6) 1951.)

ber of clinical phenomena heretofore attributed to branching of nerve fibers or even to central effects.[13, 14]

Most often this mechanism has been suggested as the basis of certain sensory phenomena, such as paraesthesia, hyperpathia, causalgia and so on.[13, 14, 20] Indeed, it has been demonstrated experimentally that sensory fibers are more susceptible to spontaneous discharge and fiber interaction than are motor fibers, although the difference is only one of degree.[13]

There is no conclusive evidence that the mechanism just described is the mechanism of hemifacial spasm. It is only true that, thus far, our observations have not been inconsistent with this concept. The relief of hemifacial spasm by decompression of the facial nerve suggests that the genesis of the synkinesis and spasm may lie in the facial canal. The observation of a dense fibrous nodule in 1 patient and of a thickened sheath of the nerve in the facial canal in others suggests that injury of the nerve may occur by compression of the nerve and its blood supply.

Electromyographic studies of hemifacial spasm have revealed patterns of action of motor units in the facial muscle which resemble those associated with irritant lesions of nerve trunks which have been observed clinically[21] or have been produced experimentally.[14] One type of spontaneous activity is illustrated in Figure 2. This activity is a grouped electrical discharge produced by rapid repetitive action of a motor unit. Action potentials of this type are not seen in normal muscle, but are often observed in muscles when the motor nerve becomes involved in an irritative process. In a patient with Bell's palsy who had been operated on, it was found that a red, dense, fibrous collar encircled the nerve at the stylomastoid foramen and that the nerve itself was greatly enlarged, red and congested near the foramen. If we may draw any conclusion therefrom, it would be that in these cases the pathologic changes visible under low magnification were essentially the same in the presence of both disorders. Other surgeons who have uncovered the nerve in cases of Bell's palsy have described similar changes. Morris[22] found a series of nodules that resembled small neurofibromas along the course of a white shrunken nerve; Cawthorne[23] found a constricted and hemorrhagic nerve at the stylomastoid foramen, and, in 2 other cases of long-standing palsy, shrunken, reddened nerves that were adherent to the sheath. Bergström[24] found a constricted facial canal; for practical reasons it should be noted that he reported a case in which excellent regeneration followed decompression of a nerve that had been completely paralyzed for fifteen years. Kettel[18] found necrosis of the cellular system and exudation. In 3 of Kettel's[18] patients he observed that decompression of the nerve at once relieved an assumed facial contracture. Lest the wrong impression be given, it should be noted carefully that Campbell and Keedy[25] described circoid aneurysms in the cerebellopontine angle in 2 cases of hemifacial spasm associated with trigeminal neuralgia, and that tumors in the

same location have been associated with hemifacial spasm. Although such cases are not common, the patient should be examined with such possibilities in mind. From what has gone before, it is more likely that the precipitating cause of hemifacial spasm is to be sought in the facial canal, where encroachment on the limited space results in compression of the nerve and its blood supply. The clinical expression of this is a tunnel syndrome.

Of particular interest in our patients was the recovery from the synkinesis. When such recovery occurs within a few days, or if it occurs at all, it can only mean that some explanation other than misdirection or branching of nerve fibers must be sought. An explanation suggested by Bickford and one of us (Lambert[26]), namely, that of "cross firing," or the excitation of fibers by impulses traveling over adjacent fibers, appealed to us because of its simplicity, clinical fitness and at least partial experimental support.

SUMMARY AND CONCLUSIONS

The operation of neurolysis for hemifacial spasm has proved somewhat disappointing in that there is a tendency for recurrence of the spasm after a time, although most patients report considerable relief from the procedure. In 1 patient the spasm appeared to recur with recovery of the postoperative facial palsy. In others it occurred after a delay; 2 patients reported no return in nine months. Of the first 2 patients operated on, 1 had recurrence of about 50 per cent of the hemifacial spasm after a year and a half, but he expressed himself as pleased with the result. In the other, after a year, the spasm returned in full force and in addition there remained some disturbance of taste, probably caused by injury to the chorda tympani in its recurrent course. Of the remaining 4 patients, 2 were free of hemifacial spasm after nine months. It is considered that this may be too short a time to permit estimation of the ultimate result. Of the other 2 patients, 1 had a long-lasting facial weakness which had not entirely abated at the time of this report; the second had not yet sent us a report when this paper was written.

In the patient with persistent paralysis, the sheath of the nerve was distended by injection with isotonic solution of sodium chloride before slitting. Whether this distension had an influence on the duration of the palsy is not known.

The delay in return of the spasm is of major interest in these patients, because this delay suggests that the hypothesis of the dichotomy of axons as a cause of spasm, synkinesis and contracture may not be correct. It would seem probable that if axon dichotomy were the cause of this symptom, in the first place more evidence of injury to the nerve would be present than is found in patients with hemifacial spasm, and, second, if axon dichotomy were the explanation of associated movements or synkinesis in these patients, it would seem that these signs should return immediately with the return of function. It might be argued, however, that injured nerve fibers are more vulnerable to additional injury, and that if only a few axons were involved in this process, these might be destroyed by the trauma of surgery, or at least might be the last to recover.

While the operation has been somewhat disappointing from the clinical standpoint, the results of the procedure have invited a certain amount of speculation about the mechanism of hemifacial spasm.

MAYO CLINIC.

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DISCUSSIONS

DR. JOSEPH A. SULLIVAN: Dr. Williams has presented a subject that has been a matter of a great deal of concern for many years, much more to the neurologist and the neurosurgeon than it has to the otologic surgeon.

However, for purposes of record, the first case of hemifacial spasm that I subjected to this operative procedure was 16 years ago. At that time I decompressed the nerve and slit the neurolemmal connective tissue sheath. Much to my chagrin within two or three months the hemifacial spasm recurred worse than before surgery. Therefore I did not operate again for relief of hemifacial spasm for three years. During this period of time we were intensely interested in the

occurrence of facial spasm following the nerve grafting operation. Over a period of years it has been our opinion that there was less likelihood for the facial spasm to develop in a nerve that had been injured when (1) Subjected to facial repair immediately, say within seventy-two hours; (2) Had undergone degeneration for a period of 6 months before repair was undertaken, than (3) One that was repaired 2 or 3 months following injury.

Dr. Bast, yesterday in his very able discussion of Dr. Shambaugh's paper, made this important statement, "Physiologic disturbances produced by operation give rise to an increased vascularity, and take care of this disorder for the time being." By that I do not mean in any way whatsoever to contradict what Dr. Williams has stated. I have used this procedure myself in highly selected cases and their selection is difficult.

I might ask Dr. Williams how he differentiates a tic from this condition?

In one of his interesting paragraphs he used the word "dichotomy"—and Dr. Fenton can correct me—which comes from two Greek words meaning to cut and twofold. The delay in the return of the spasm suggests that the hypothesis of the dichotomy of axons as a cause of spasm, synkinesis and contracture may not be correct. That leaves the question entirely open.

These cases are just as much a psychological problem as the ones with complete facial paralysis, and they expect to have something done if at all possible.

This is the only procedure to date that in any way has alleviated their complaint for a period of time. I wonder if Dr. Williams has any explanation of why in the case where he injected normal saline into the sheath that the paralysis still has persisted.

In closing my remarks, I would like to state this, that we feel that this procedure is indicated in certain cases. They are very difficult to select, the cause of the condition is still an open question, and I can only read from the staff meetings of the Mayo Clinic, June 1951, in their concluding paragraph when they state, "the facial canal is probably not the only site of the tunnel syndrome, it would seem proper to inquire anew the nature of certain other clinical phenomena, not only of peripheral but of *central* origin for which current explanations are not quite satisfying."

Dr. Frank D. Lathrop: It is interesting that in the past, this condition which is most distressing, has been taken care of by the neurosurgeon. At the present time the operative procedure of choice is selective nerve section of the branches of the facial nerve at the periphery that appear to be most productive of the spasm. This has proven to be quite satisfactory if the section is carefully performed. It is a very minor procedure and will give rise to perfect relief, although there will always be an associated paralysis of that portion of the anatomy of the face which was innervated by that branch of the facial nerve.

In each of Dr. Williams' cases there has been evidence ot some injury to the facial nerve following the decompression. It is my belief that it is the injury to the facial nerve which has resulted in minor temporary depression of function that affords the patient the relief that he obtains from the hemifacial spasm. The facial paralysis will always disappear I am quite certain and perhaps the hemifacial spasm will disappear permanently in sufficient number of patients to make this procedure become the procedure of choice in the treatment of this most distressing condition. Even if it does not entirely relieve the hemifacial spasm, I believe there will be sufficient improvement in carefully selected cases so that we can

take the treatment of this condition into our own field of practice and take it away from the neurosurgeons, which would make me most happy.

DR. M. F. MCCARTHY: If I had discussed this paper a year ago, I would be more enthusiastic than today. We heard by the grapevine that this work was being done at the Mayo Clinic.

(Film) A man about 33 years of age was having great difficulty. The girls in the street thought he was winking at them and he was getting into a great many difficulties. He has a slight amount of facial weakness.

A week after the operation the twitching had practically disappeared. The man joined the Army shortly after and we were able to get in touch with him about a month ago and he tells us he was completely free of spasm until four months ago. He is satisfied with his operation because the spasm is not so marked as it was previously.

The other patient we have operated upon is now living in Bermuda. The last communication I had from her indicated that she was satisfied.

Roughly we can say our results are 50 per cent.

DR. SAMUEL ROSEN: I should like to report on a case similar in some respects to those reported here by Dr. Williams. This woman about 55 had a very marked right hemifacial spasm. However, through previous disease she was totally deaf in her left ear. It therefore seemed a little bit dangerous to decompress the facial nerve since I heard from Dr. Williams that sometimes the hearing is diminished after this operation. This woman had marked tinnitus on the side of the spasm and also had marked hypergustatory sensations on the anterior two-thirds of the same side of the tongue. Because stimulating the chorda tympani gives rise to this tic-like contraction of the face, I tried sectioning the chorda tympani nerve instead of decompressing the facial nerve. Following the operation the spasm was still present but it was about 50 to 75 per cent less than what it was before the operation. After five or six months she still feels pleased. The tinnitus on that side disappeared after cutting the chorda tympani nerve.

I would also like to relate one other case that I saw many months ago in Israel. I was asked to see a patient there who had a typical Meniere's disease of several years' duration, a man about 45 years old. He had a marked hemifacial spasm on the side of the disease and I wondered if there might not be some connection between the function of the chorda tympani nerve, the syndrome of Meniere and hemifacial spasm.

DR. HENRY L. WILLIAMS: I am unable to answer Dr. Sullivan's question about why paralysis persisted so long after the injection of saline was done, in view of my lack of experience. I was trying to blame it on the technic of injection but the neurosurgeons say that is unfair, that the injection itself should not produce a long-lasting paralysis.

I will certainly have to investigate the surgery of the chordatympani. It seems to be developing into a surgical panacea and we may not need any other type of surgical procedure for our operations on the ear.

LXXIV

DEVELOPMENTAL ANOMALIES OF THE SOUND CONDUCTING APPARATUS AND THEIR SURGICAL CORRECTION

GEORGE E. SHAMBAUGH, JR. M.D.

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Developmental anomalies of the sound conducting apparatus, while not so very common, are of particular interest to the patient and to the otologist. They are important to the patient because of the hearing impairment from birth which, if bilateral, may be sufficient to interfere with the normal development of speech. The patient is also concerned by the cosmetic appearance when an external deformity accompanies the anomaly of the sound conducting system.

The otologist finds these anomalies interesting because they reflect the phylogenetic and embryologic origin of the hearing mechanism, and because they are often correctible with a useful improvement in hearing.

The prevailing attitude toward surgical correction of congenital atresia was generally pessimistic[1] despite occasional reports of successful operations by Page,[2] Dean[3] and a few others, until 1947. In that year Pattee[4] in the United States and Ombredanne[5] in France each reported a series of cases successfully operated to improve the hearing. Other articles by these authors,[6, 7] and by others[8, 9, 10] have followed.

Both Pattee and Ombredanne construct a new external auditory meatus down to the middle ear cavity, but Pattee relies on a skin graft against the middle ear cavity and stapes, to act as a substitute tympanic membrane and thus to improve the hearing, whereas Ombredanne in addition fenestrates the horizontal semicircular canal. Thus the literature leaves some doubt as to the best procedure for these cases.

In order to understand the possible anomalies of the ear that may be encountered and to effectively deal with them, it is necessary to have a clear idea of the embryologic development of the ear.

The two principal divisions of the organ of hearing come from different embryonic anlages. The sensory end-organ in the inner ear comes from the ectodermal *otocyst,* while the sound conducting apparatus in the outer and middle ear comes from the *gill structures.*

The development of the endolymphatic labyrinth from the otocyst is not the main concern of this discussion, but a few points will be mentioned to contrast it with the development of the sound conducting mechanism.

The membranous labyrinth has its first beginning in the 3½ week old human embryo[11] as a thickening of ectoderm on either side of the head: the otic placode. This area infolds to become the otic pit, then pinches off to become the otocyst. By means of a series of folds the otocyst differentiates in the three month foetus into the endolymphatic duct and sac, the semicircular endolymphatic ducts, the utricle, saccule, and cochlear duct containing the organ of Corti. It is interesting to reflect that our "fishy ancestors" swam in seas not yet as salty as today's ocean, and that endolymph closely resembles in chemical composition the dilute salt water of that primeval sea, entrapped by the infolding otocyst.

By the fifth month of foetal life the sensory end-organ of the ear attains adult form and size as the cartilaginous otic capsule ossifies. Thus in a certain sense the inner ear has already begun to age by the time the infant is born. This might help to explain some cases of progressive perceptive deafness in early and middle adult life as instances of premature presbyacusis.

Our ancient aquatic forbears did not need any special mechanism to transmit sound to the inner ear. As in today's fish, sound was readily transmitted from the sea through the skin to the fluid of the inner ear. But when these enterprising ancestors of ours struggled out of the seas onto dry land a new problem presented: 99.9% of the energy of air-born sound is reflected away from the surface of sea water,[12] due to the different physical characteristics of the two mediums. To overcome this sound barrier a mechanical device was needed to convert air vibrations of large amplitude and small force into fluid vibrations of small amplitude and large force. The

gill structures, no longer needed for breathing, become converted into such a mechanism. The first branchial groove became the external auditory meatus while the first pharyngeal pouch became the eustachian tube and middle ear. Then, instead of the branchial groove and pharyngeal pouch connecting to become a gill cleft, a thin layer of tissue remained between to form the tympanic membrane. From the cartilage of the first branchial arch (Meckel's cartilage) came the mandible, incus and malleus, while the stapes, styloid process and hyoid bone came from the cartilage of the second arch (Reichert's cartilage). The large area of the tympanic membrane connected by the lever-system of the ossicular chain to the small area of the oval window, gave the ear an effective impedance-matching mechanism to overcome the sound barrier between air and water. Deprived of this mechanical apparatus by interrupting the ossicular chain, the ear loses from 20 to 30 decibels of hearing. This is why the successfully fenestrated ear has a residual conductive loss averaging about 25 decibels for the speech frequencies.[13]

The gill structures destined to form the sound conducting apparatus first appear in the human embryo at 4 weeks, at about the same age as the otic pit. By the third foetal month the pinna has been formed from the first and second branchial arches on either side of the first branchial groove which is the primary shallow funnel-shaped external auditory meatus. From the inner end of this primary meatus a solid cord of ectodermal cells extends inward, with a bulb-like enlargement adjacent to the middle ear. Not until the seventh foetal month does this cord canalize, beginning medially to form the tympanic membrane and then extending laterally to join with the primary meatus to form the completed external auditory meatus. The outer and middle ear, now ready to transmit sound to the inner ear, are not yet of adult form or size. Pneumatization of the temporal bone by mucous-membrane lined excavations extending out from the middle ear cavity also begins in the seventh foetal month, while the sub-epithelial jelly-like mesodermal tissue in the middle ear cavity begins to resorb. At birth the eustachian tube inflates, the sub-epithelial connective tissue in the middle ear attic and antrum continues to resorb until the epithelium lies close to the periosteum, and pneumatization of the temporal bone proceeds.

The external auditory meatus, entirely cartilaginous at birth except for the narrow incomplete ring of the tympanic bone, deepens by growth of the tympanic bone to form the adult osseous meatus.

Except for some pneumatization of the petrous apex that may continue into adult life, the outer and middle ear finally reach adult form and size in late childhood, in contrast to the inner ear that became adult in mid-foetal life.

Since the two principal divisions of the organ of hearing come from different embryonic anlages, there is a marked tendency for anomalies of the inner ear to be independent of anomalies of the outer and middle ear. Of the 22 ears with developmental anomalies of the sound conducting apparatus operated by the writer and his associates, 17 had normal cochlear function, 2 certainly had a cochlear defect, presumably developmental, and 3 probably had a cochlear defect, but are still too young to permit reliable bone conduction testing.

Congenital anomalies of the inner ear occur during the second or third month of embronic life, before differentiation of the endolymphatic duct system is complete. Congenital nerve deafness from rubella in the mother in the first trimester of pregnancy is a familiar example.

Congenital anomalies of the sound conducting apparatus occur during the second to the seventh month of foetal life, the particular deformity in each case depending upon the age of arrested development and the area of the gill structures chiefly involved.

The commonest anomaly in our series, and according to the literature, is the result of failure of the ectodermal cord to canalize. The pinna may be absent, microtic, or it may be normal. The external auditory meatus may be absent, or consist of a shallow funnel-shaped blind depression, beyond which lies an irregular mass of fibrous tissue cartilage and bone, with a bony plate at the site for the tympanic membrane. The malleus and incus may be normal or deformed, but the stapes is usually normally formed and mobile.

Less common is a partial canalization of the ectodermal cord, with a formed tympanic membrane but an atresia between the inner end of the meatus and the primary meatus. This was present in 5 of our cases.

Sometimes the developmental anomaly is chiefly of Reichert's cartilage with deformity and fixation of the stapes. Altmann[9] described the histologic findings of a case of atresia with stapes fixation. Five of our operated ears had a patent meatus, and intact tympanic

membrane, but a congenital stapes fixation. This condition has not been described previously in the literature to the writer's knowledge, with the exception of Altmann's histologic study. Congenital stapes fixation may be differentiated from otosclerotic stapes ankylosis and may be diagnosed during life by the following characteristics:

1. There may be an atresia of the opposite ear, suggesting a possible anomaly of the stapes.

2. The deafness dates from birth.

3. The hearing loss is not progressive.

4. There is no family history of otosclerosis.

5. At operation there is no visible otosclerotic focus at the oval window, the stapes and its footplate are deformed, and other structures in the middle ear may be malformed.

Not infrequently anomalies of the sound conducting apparatus are accompanied by aplasia of the mandible (which comes from Meckel's cartilage of the first branchial arch) with an "Andy Gump" profile. Occasionally in atresia there is a congenital facial nerve paresis. If there is an aplasia of the maxilla along with the atresia the patient has the Treacher Collins syndrome, with notching of the outer third of the lower lid and downward sloping outer angle of the eyes.

Congenital atresia, according to the literature, is unilateral more often than bilateral and on the right side more than the left, although these differences were not brought out in our small series in which 7 patients were unilateral and 8 had a bilateral atresia, while 4 of the unilateral atresias were of the right ear; 3 of the left ear.

INDICATIONS FOR CORRECTION

Pattee[6] states that all bilateral cases of congenital atresia of the auditory canal should be operated on in an attempt to improve the hearing. To this we can add that normal cochlear function and good pneumatization are desirable but not absolute requisites, provided there is not a profound cochlear defect.

At first we advised against operation in unilateral cases, but with greater confidence from experience we believe that unilateral cases may be benefited enough to justify surgery, both cosmetically and

in the hearing improvement that permits better localization of sound even though the operated ear does not reach the hearing level of the normal ear.

The optimum age for correction for bilateral cases is as young as adequate cochlear function can be determined, which is probably not before the age of 4. In unilateral cases there is less urgency and operation had best be delayed until the child is old enough to better tolerate surgery and the post-operative care.

TECHNIC FOR SURGICAL CORRECTION

Since the particular anomaly in each case varies greatly, no one technic should be suitable for all cases. In each case the surgical procedure should be fitted to the condition that is found, keeping in mind that the aim is to construct a sound conducting system that will most nearly approach the normal impedance-matching mechanism.

The most favorable cases are those with an intact tympanic membrane and mobile ossicular chain beyond the atresia. One need only establish a patent meatus, lined by skin grafts to create an essentially normal impedance-matching sound conducting apparatus, with hearing that may approach or attain normal.

If there is no tympanic membrane, but normally formed and mobile ossicles, we should expect the best result from placing a skin graft against the malleus and incus as a substitute tympanic membrane. In some cases subsequent cicatrization may fix these ossicles, in which case they should be removed in a second operation. If the stapes becomes fixed by subsequent cicatrization fenestration of the semicircular canal is indicated. If there is a congenital stapes fixation fenestration is indicated at the first operation. We do not believe that it is wise to fenestrate all cases routinely as advocated by Ombredanne: It is not necessary and it opens the labyrinth with the risk of possible serous labyrinthitis leading to permanent cochlear damage.

The end-aural approach is the most direct and safest for correction of these anomalies. If there is a shallow funnel-shaped primary meatus the usual end-aural incision may be used. If there is no meatus, a crucial incision as described by Page[1] will enable one to form a round meatus with smoothly incurved edges.

The region of the normal osseous meatus is carefully exposed with the mastoid process and root of the zygoma. If a mass of tissue fills the area of the normal osseous meatus, it should be carefully removed along with some of the posterior and superior osseous meatal wall, to see whether an intact tympanic membrane might exist. If so, it is better to avoid opening the attic if possible, the new osseous meatus being lined by Thiersch split thickness skin grafts. In many cases, however, the head of the mandible lies against the mastoid process, and the middle ear must be opened via the attic. Always keep in mind the possibility of an aberrant facial nerve when operating on anomalies of the ear. Facial nerve paralysis resulted from the first recorded operation for atresia by Kiesselbach in 1883, and it has occurred in a number of recent cases, reported and unreported. In one of our cases an attempt to correct the atresia elsewhere had resulted in a facial nerve paralysis.

Sometimes the anomaly includes a much reduced or even absent antrum, attic and tympanic cavity. The risk of severing the facial nerve while searching for an antrum that does not exist is particularly great. Therefore, we prefer the atticotomy approach as used in fenestration surgery. If the attic is not found at the usual site it may be prudent to terminate the operation rather than risk adding a facial paralysis to the existing congenital deformity.

As soon as the attic and middle ear are exposed the type and extent of the anomaly is determined by careful inspection under magnification, and the appropriate corrective procedure is selected, aiming to construct a sound conducting apparatus that will most nearly approach the normal in its impedance-matching action.

RESULTS OF CORRECTION OF ANOMALIES OF THE SOUND CONDUCTING APPARATUS

Of the 22 ears operated by the writer and his associates, (1 by Arthur L. Juers, a former associate, 7 by Eugene L. Derlacki, and 14 by the author) 1 consisted of a facial nerve repair of a case unsuccessfully operated elsewhere, and in 1 case the operation was terminated when the attic was not found. Of the 20 operations completed for improvement of the hearing, all received an initial hearing gain. In 2 the gain was not maintained: 1 was lost due to osseous closure of the fenestra in the semicircular canal, 1 was lost due to fibrosis fixing the ossicles. Of the 18 ears with maintained hearing improvements, 1 was revised with skin grafting of the meatus

TABLE 1.

Case	Ear	Age At Op.	Meatus	M.T.	Malleus & Incus	Stapes	Surgical Procedure	Cochlear Function	Pre-op Loss (512, 1024, 2048)	Last Post-op Test DB Loss	Time Post-Op.
1 B	L	7	Atresia	Normal	Normal	Normal	Cr. Meat. with Graft	Normal	56 db	18 db	5 yrs.
	R	7	Atresia	Normal	Normal	Normal	Cr. Meat. with Graft	Normal	50 db	17 db	5 yrs.
2 B	R	7	Patent	Normal	Normal	Immobile	Fenestration	Normal	52 db	27 db	3 yrs.
	L	10	Patent	Normal	Normal	Immobile	Fenestration	Normal	58 db	23 db	6 mos.
3 B	R	6	Facial Nerve Repair						62 db		
4 B	R	4	Atresia	Present	Sl. Def.	Normal	Cr. Meat. with Graft	? Imp.	?	63 db	3 yrs.
	L	5	Atresia	V. Thick	Def.	Normal	Cr. Meat. with Graft	? Imp.	?	52 db	2 yrs.
5 B	L	7	Narrow	Normal	Sl. Def.	Def.	Fenestration	Normal	59 db	30 db	3 yrs.
	R	10	Narrow	Large	Cholesteatoma	M.E.	Enlarg. Meat.	Normal	60 db	50 db	2 wks.
6 U	R	18	Atresia	Absent	Normal	Normal	Cr. Meat. with Graft	Normal	55 db	25 db	2½ yrs.
7 B	L	15	Atresia	Absent	Def.	Normal	Cr. Meat. with Graft, Ossic., Fenes.	Imp.	97 db	77 db	2½ yrs.
8 U	R	22	Atresia	Absent	Def.	Normal	Cr. Meat. with Graft, Ossic., Fenes.	Imp.	95 db	55 db	2½ yrs.
9 U	L	13	Atresia	Absent	Normal	Normal	Cr. Meat. with Graft, Revised 3 mos. Graft	Normal	58 db	23 db	2½ yrs.
10U	L	24	Atresia	Small	Normal	Normal	Cr. Meat. with Graft	Normal	36 db	? 3 db	2 yrs.

11B	R	25	Normal	Normal	Normal	Immobile	Fenestration	Normal	47 db	17 db	20 mos.
12B	L	25	Atresia	Absent	Def.	Normal	Cr. Meat. with Graft, Ossic.	Normal	68 db	25 db	7 mos.
13U	L	5	Atresia	Absent	Def.	Normal	Cr. Meat. with Graft, Revised with Ossic.	? Imp.	?85 db	53 db	10 mos.
14U	R	9	Atresia	Absent	Def.	Normal	Cr. Meat. with Graft, Ossic.	Normal	73 db	17 db	1 yr.
15B	R	14	Normal	Normal	Normal	Immobile	Fenestration	Normal	48 db	42 db	1 yr.
16U	R	17	Atresia	Antrum & Attic not found			Op. not completed	Normal	75 db		
17B	L	11	Atresia	Absent	Sl. Def.	Normal	Cr. Meat. with Graft	Normal	41 db	42 db	9 mos.
18B	R	4½	Atresia	Absent	Def.	Normal	Cr. Meat. with Graft & Ossic.	Normal	53 db	32 db	6 mos.

after a secondary atresia, 1 was revised with removal of the incus and malleus due to secondary fibrosis and fixation. Both of these regained their improvement and have maintained it.

Of the 22 operated ears with an anomaly of the sound conducting apparatus, 15 had an atresia, 5 of these with a formed tympanic membrane, 10 with a bony plate replacing the tympanic membrane. In 10 of the 22 ears the malleus and incus were malformed (Meckel's cartilage), in 5 the stapes was deformed (Reichert's cartilage).

Of the 18 ears with maintained improvements in hearing, 11 are at the practical 30 db level or better at the most recent test, 7 of these having maintained this result for 2 years or longer.

CONCLUSIONS

1. Each anomaly of the sound conducting apparatus can be traced to arrest of development of a particular area of the gill structures at a particular age of foetal life.

2. The most frequent anomaly is atresia due to failure of the ectodermal cord to canalize in the 7th foetal month. In some canalization began but was not completed with a formed tympanic membrane and atresia. There are the most favorable for surgical correction.

3. The majority of anomalies of the outer and middle ear have normal cochlear function.

4. Anomaly of the stapes with congenital stapes fixation can be differentiated from otosclerotic stapes ankylosis by:

a). A deformity of the outer or middle ear on the opposite side suggesting a congenital defect.

b). The hearing impairment dates from birth.

c). The hearing impairment is not progressive.

d). There is no family history of otosclerosis.

e). At operation the stapes is seen to be deformed, and there is no otosclerotic focus at the oval window. Other malformations of the middle ear may be observed.

5. No one surgical technic is suitable for all cases. In each case the procedure should be modified according to the deformity, the aim being to construct a sound conducting apparatus most nearly approaching the normal impedance-matching mechanism.

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DISCUSSION

DR. THEODORE E. WALSH: I would like to say one or two things about Dr. Shambaugh's results. We have operated I think on only five congenitally malformed ears. In one case I was particularly interested in leaving the incus and malleus as they were perfectly movable on either side. Neither side had a drum but had a bony partition instead. On one side I removed the incus and malleus and placed a Thiersch graft, not full thickness. The hearing on that side has come up I think in terms of social adequacy to 63, which is very adequate. On the other side having left the incus and malleus I got very little improvement. It is quite inadequate. I wonder if Dr. Shambaugh would not say something about the comparative value of the Thiersch to the full thickness graft. I have been a little afraid to use the full thickness graft but I understand from conversations around it is a very good method. I would like a litle information.

Apropos of fenestration, I agree with Dr. Shambaugh about the inadvisability of doing the fenestration at the same time as you do this reparative operation. In the case in which we took out the incus and malleus we got perfectly adequate hearing and I am certain that the patient could not be further improved by fenestration because there is no further that you can go. The limit of improvement through fenestration as you heard is about to the 20-25 db level owing to the disruption of the ossicular chain. Fenestration could not help this patient fur-

ther. It would have been an awful risk to have done it at the same time. I cannot go along with the French author on this score. However, in some cases in which there has not been sufficient improvement in hearing from the graft—and I have had one woman in whom we did both ears—we fenestrated in a subsequent operation and I think had about a 25 db gain from the fenestration over what we had before. Following the first operation she still needed a bone conduction hearing aid. Since the fenestration she has been able to get rid of the aid and hears perfectly well for ordinary conversation.

DR. CHARLES E. KINNEY: There is one thing that I would like to ask Dr. Shambaugh's opinion about, and that is the use of the term "atresia." Personally I prefer to confine the use of the word to those cases in which there has been complete or nearly complete canalization of the external auditory meatus and subsequently either due to surgery, accident, burn or other cause a true atresia developed. He has explained this very beautifully in the movies but still calls it atresia. I wonder if it would not be better to use the term "aplasia."

Another comment that I would like to make having done four of these, is that I can testify they tax one's surgical ability and certainly one's surgical fortitude. Two of the four cases on which I have operated were rather severe types of Treacher-Collins' syndrome and I would like to mention that in both cases the temporalis muscle completely covered the temporal bone which was a disturbing situation to handle. In the first of the two that I did, the child was 35 months old and I was keeping in mind an admonition by my teachers about the exit of the facial nerve in a young child being quite high up. I was particularly careful of the lower end of my original incision. Immediately upon recovery from the anesthesia in this case there was, much to my astonishment, a complete facial paralysis on that side. We reoperated that patient on about the 19th post-operative day because of this post-operative paralysis and because the skin graft which we had used had not taken nearly as well as it had in my two previous cases. Much to my surprise in re-operation we discovered that the facial nerve had emerged from the temporal bone approximately one centimeter cephalad to where we located the middle ear and I had cut th facial nerve at the *upper end* of my endaural incision. I reported this case last fall but we had only operated her three months before. It is now approaching ten months and I am happy to report that this little girl has about 85 per cent recovery of the facial movements on that side. We simply did an end-to-end anastomosis of the severed nerve. The point being, in these abnormally developed children this facial nerve can be, as Dr. Shambaugh pointed out, in very queer places and some of our old admonitions have to be revised.

DR. GEORGE L. PATTEE: All patients on whom I have operated had complete bony occlusion of the canal and I have not had any patients with a membrana tympani. The deformity is not the same in every case and I am sure that in those cases with the least deformity of the ossicles, you are apt to get better postoperative results. It is important that you don't injure the facial nerve, as Dr. Kinney and Dr. Shambaugh told you.

An unusual thing happened to one of my patients postoperatively. This boy lived in Kansas and not until he was eleven years old did an examination of the ear reveal that he had complete closure of the auditory canal. He was operated on and had a good improvement in hearing according to my audiograms. The following fall the mother asked his teacher whether she had noticed any change in his hearing. This teacher had had no training with the deaf, no knowledge of the sound of the voice of the deafened person, and she told the mother that

she did not think he heard any better than before, but that he now corrected her pronunciation.

In view of the fact that we were taught for so many years to let these cases alone, I am glad to see otologists approve and forward the work on this problem.

We are often accused of getting our medicine from the lay magazines. Some time ago in Time Magazine there was an article entitled "The Redheads and Blackbottoms," about an archeological expedition from the Santa Barbara Museum of Natural History. It told of their findings in two burial grounds on the islands off Santa Barbara. Due to the method of embalming used, all the skeletons in one burying ground had red heads, and in the other one, black pelves, hence the name, "Redheads and Blackbottoms." The thing that caught my attention was that a large percentage of the skulls showed bony occlusion of the auditory canal. I wrote Dr. Orr, an archeologist, of the Santa Barbara Museum and asked him about it. He could not give me the information I wanted. They had cleaned up several skulls and set me photographs of them.

From the pictures I cannot be sure whether there was exostosis or bony atresia, but they had cleaned up only twelve skulls and four of them showed bilateral bony occlusion. I hope some of my California friends will go up to Santa Barbara and look at these skulls and see whether the closures are due to exostosis or to congenital malformations.

I asked Dr. Orr whether the large numbers found in one burying ground might be due to inbreeding. He said he did not think so as they probably were no more inbred than the other Indian tribes. If these are congenital malformations and found so frequently in one burying ground, it is quite unusual.

DR. HARRY C. ROSENBERGER: I would like to have Dr. Shambaugh discuss, if he will, the subject of wether cases of unilateral congenital malformation of the external auditory canal should be operated on.

DR. HOWARD P. HOUSE: The importance of hearing in these children is now being recognized both by pediatricians and plastic surgeons. In the past the primary concern seemed to be with the cosmetic problem rather than with the hearing impairment. Consequently, we will be called upon more in the future for our advice than has been prevalent in the past.

In my opinion, surgery for the unilateral case may well be delayed until adulthood and cosmetic concern may take precedence. In bilateral cases, however, the hearing urgency should take precedence over the cosmetic concern.

The optimum age for surgery, in my experience, has been four years. At this age we are able to determine the presence of adequate cochlear nerve reserve and can determine the presence of a middle ear by x-ray studies, as suggested by Gilbert Roy Owen. In the pneumatized case the prognosis is much better than in the sclerotic patient. Fortunately, pneumatization occurs more frequently than sclerosis.

During the first four years every effort should be made to develop sound awareness, and speech training. This can be done through parent cooperation and amplification.

The maximum hearing improvement, in my opinion, is obtained by leaving the bony ear drum intact and making the smallest possible opening into the middle ear for the purpose of removing the deformed incus and malleus head. A full thickness graft is placed over this opening and apparently mucous membrane from the middle ear lines its under surface. This allows the creation of a middle ear

air space and eliminates the problem of the graft lying immediately over the stapes. If practical hearing is not obtained by this means, then fenestration may be done as a secondary procedure, or as a primary procedure if the stapes is found to be fixed at the time of the initial operation.

I routinely place an acrylic mold in the ear canal three weeks after surgery in order to prevent postoperative stenosis.

DR. GEORGE E. SHAMBAUGH: Dr. Walsh brought up the problem of whether the incus and malleus should be removed in all of these cases. It has been our feeling that we could create a sound conducting mechanism more nearly approaching normal if a normal and mobile incus and malleus were left in. However, with experience we may change our mind although from cases thus far operated this seems to be a satisfactory procedure with the possibility of removing these ossicles at a second operation should they become fixed through secondary cicatricial changes during healing.

Dr. Walsh asks also about a full thickness graft as compared to a Thiersch graft for constructing a substitute tympanic membrane. We have used a full thickness graft from behind the ear made as thin as possible for the substitute tympanic membrane to close off the middle ear cavity, with Thiersch grafts to line the remainder of the ear canal. The packing is left in place for two full weeks—the first five days of which the patient remains in the hospital under penicillin. When the packs are removed at the end of two weeks, the skin grafts are firmly adherent.

Dr. Kinney objects to the word "atresia," since it implies that there was once a canal which subsequently closed. If he will supply a suitable simple word to take its place, I will welcome it.

The malposition of the facial nerve in these cases is very disconcerting, and its possibility must always be kept in mind. In one of our patients with a patent meatus having a congenital anomaly with fixation of the stapes, the tympanic portion of the facial nerve ran in an almost vertical direction with distortion of the medial wall of the middle ear.

Dr. Rosenberger brings up the question of operating on unilateral cases of congenital atresia. Initially, we did not believe that unilateral cases required operation. Then we were sent several patients by a plastic surgon who wished to have the meatus constructed before he began to try to create an auricle. One of these cases was operated on, and the affected ear brought from a 60 decibel loss to a 25 decibel level. This permitted the patient to localize sound for the first time and she has been quite pleased with this improvement in hearing. We now believe that operation in unilateral cases might be justified for the improvement in hearing as well as to help the plastic surgeon in his subsequent work.

Dr. House mentioned the cosmetic prostheses to improve the appearance of the ear. We certainly have not been particularly impressed with the results of plastic surgery to create a new auricle as most of these result in a cauliflower-like nubbin on the side of the head. He brings up the age for correcting these congenital anomalies of the ear. In the bilateral cases we think the younger the better, at least for one side, since it helps with the normal acquisition of speech. Our youngest patient was operated on at the age of 4. It was not possible to obtain accurate audiometry on this patient, but we did determine that he had fairly good bone conducted hearing. This child at the time of his operation could not speak a single word. Within two weeks after operation he began to

talk and learned speech quite rapidly once a patent meatus had been established with improved hearing. Normal cochlear function is desirable but not absolutely necessary provided the cochlear defect is not profound. Four of our patients had a partial cochlear defect with an anomaly of the outer and middle ear, yet the improvement in hearing which they experienced was very worthwhile, although the hearing is still far from the practical level.

Time did not permit a detailed description of the surgical technic for correcting these anomalies. Dr. House has a beautiful motion picture of the technic.

There is one point I would mention: In the normal ear the articular process of the mandible is separated from the mastoid process by the osseous meatus. In congenital atresia there may be a mass of fibrous tissue and bone where the external auditory meatus should be, and in other cases the head of the mandible lies immediately against the mastoid process. In these we use the atticotomy approach following the middle fossa dura into the attic to avoid injury to a misplaced facial nerve.

LXXV

THE AUDIOMETER WEBER TEST AS A MEANS OF DETERMINING THE NEED FOR, AND TYPE OF, MASKING

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Difficulty is often encountered in the use of the differential air and bone conduction measurements called "the functional tests of hearing." With the theoretical exception of psychogalvanic skin response techniques, the voluntary responses of the patient are always prerequisite to the satisfactory evaluation of auditory acuity. Any confusion, misinterpretation, or misunderstanding on the part of the patient has a strong tendency to produce an inaccurate picture. Even though hearing is an autonomic activity, the ability or desire to listen and to comprehend sound must be a function of the patient. The otologist takes a history and examines the ears, nose, and throat to help him in his diagnosis; he may even obtain qualitative data from the classical tuning fork tests, but for quantitative information about the hearing, he must have audiometric measurements of both the air and bone conduction acuity of each ear.

When a significantly poorer ear is being evaluated, the better ear, for that test procedure, should be excluded. This can be accomplished by presenting noise to the better ear at an intensity great enough to "mask out," or to retrict, the function of that ear. As will be discussed anon, there are situations in which the poorer ear, for air conduction, should be masked out in order to test the function of the better ear. It is well known that in middle ear obstructive lesions, particularly otosclerosis, sound, transmitted by bone conduction, will be heard by the poorer ear.

The noise generators most generally used for masking are of the "white" and "saw-tooth" varieties, and for purposes of testing and research carried out at Columbia-Presbyterian Medical Center, these types of masking sounds have proven most satisfactory.

The most usual criteria for the employment of masking are: for air conduction, a difference of 40 decibels or more between the air conduction thresholds; and, for bone conduction, a 15 decibels difference between the air conduction thresholds—or lateralization of the bone conducted tone to the ear not being evaluated.

When testing by air conduction, the need for masking is apparent when there is a cross-over from the poorer to the better ear; this occurs when the difference between ears is more than 40 decibels. In order to eliminate the unwanted responses of the better ear, an adquate masking level must be employed. Unfortunately, however, "effective masking" can only be arrived at by probing, i.e., by increasing the masking level until there is no longer a threshold-shift in the poorer ear. Since this probing is too time-consuming to be practical in a clinical situation, and since there is always the danger of masking both ears when the masking sound is too loud, an arbitrary level is usually decided upon. On the basis of our experience, 85 decibels of either "white" or "saw-tooth" noise is satisfactory for the majority of the cases requiring masking for air conduction measurement. It must be realized, however, that there will be cases in which this level may be either too much or too little.

When testing unequal ears for bone conduction, the problem of masking becomes even more complicated. The first obstacle encountered is which ear to test first. Unless intensive study of each case has been completed prior to audiometric testing, it is the responsibility of the audiometer technician to make this decision. Utilizing the principles of the Weber Test, it should—in theory—be relatively simple to select the ear which should be tested first, and thus to which ear masking should be applied when testing for bone conduction thresholds.

The Weber phenomena proposes that a vibrating tuning fork, when placed on the midline of the patient's head, will be heard in the better ear, if the involvement is of a perceptive nature, and in the poorer ear, if the loss is of the conductive type. For the purpose described here, the bone conduction oscillator has been substituted

for tuning forks. This substitution is recommended for the following reasons:

1. The tone produced by the audiometer can be controlled in terms of both frequency and intensity,

2. The sound is sustained—that is, the tone will not "die out" as is characteristic of sound produced by forks, and

3. There is a lesser possibility of the sound being heard by air conduction.

In administering this "Audiometer Weber Test," the bone conduction oscillator should be placed on the vertex of the patient's head and, while in that position, the threshold should be obtained; 10 decibels is then added to this intensity and the patient is asked to localize the sound. The direction of localization is then recorded on the audiogram and the test is repeated for each frequency to be tested by bone conduction.

Changes in lateralization, produced by changes in the intensity of the test-tone, may indicate the presence of recruitment in the poorer ear. For example, a unilateral nerve loss will lateralize to the better ear but, if the Weber is introduced at an intensity which is loud enough, the patient will localize the sound as being at the midline; at higher intensities, the poorer ear "catches up" with the better ear and, at above thresholds, both ears are functioning equally. In certain instances, there may even be over-recruitment; the patient will then report lateralization to the better ear when low intensities are presented, and to the poorer ear when the test-tone is more intense. Unfortunately, the relatively low maximum output of the audiometer, for bone conduction, restricts the use of this recruitment test to slight and moderate losses.

If, on the Weber Test, the patient reports that he hears the sound in both ears, or in the center of the head, bone conduction thresholds for either ear may be obtained first. Otherwise, the ear to which the tone is lateralized should be first tested. Following the completion of the testing of the lateralized ear (noting at all times that the patient is certain that the sound has not transferred to the other ear), the oscillator should be placed behind the other, or non-lateralized ear, and masking introduced to the ear not being tested.

The masking levels to be used in bone conduction measurement should depend on the type of hearing loss being investigated. If the

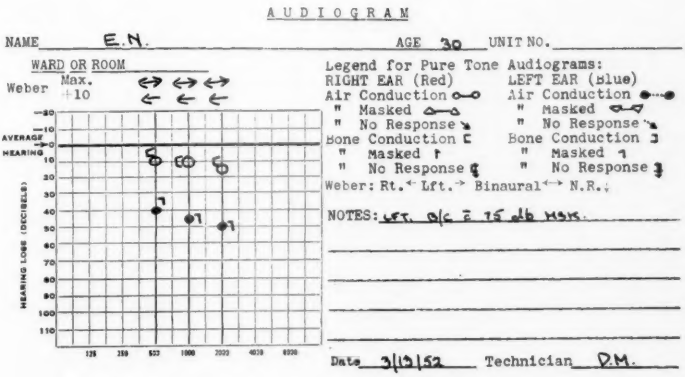


Fig. 1.—An audiogram revealing a typical unilateral perceptive loss. At 10 decibels above the midline threshold, the Weber lateralizes to the right, or better, ear; and at the maximum output of the audiometer, the tone is heard at the midline. Masking is utilized in ascertaining the bone conduction thresholds of the poorer ear.

loss is of the perceptive type, the masking level presented to the better ear, when testing the poorer ear, can be effectively arrived at by the use of Carhart's formula for "effective masking" (air conductive threshold of the better ear, minus bone conduction threshold of the better ear, plus air conduction threshold of the poorer ear). However, if the type of loss is of the conductive variety, the intensity of the masking noise presented to the poorer ear, when testing the better ear, can only be governed by the air conduction thresholds of the better ear; in order to prevent overmasking, the level of the masking noise should not exceed forty decibels above the better ear threshold of the frequency under test; if the masking level exceeds this figure, the ear under test will also be masked. For this reason, with patients who have marked unilateral conductive-type losses, the measurement of the bone conduction thresholds of the better ear cannot be obtained. Since these procedures are also too time consuming to be clinically practical, an arbitrary masking level is usually decided upon. On the basis of our experience, 75 decibels of either "white" or "saw-tooth" noise is satisfactory for the majority of the cases requiring masking for bone conduction measurement. It must be remembered, however, that there will be cases in which this level may either be too much or too little (See Figures 1 and 2).

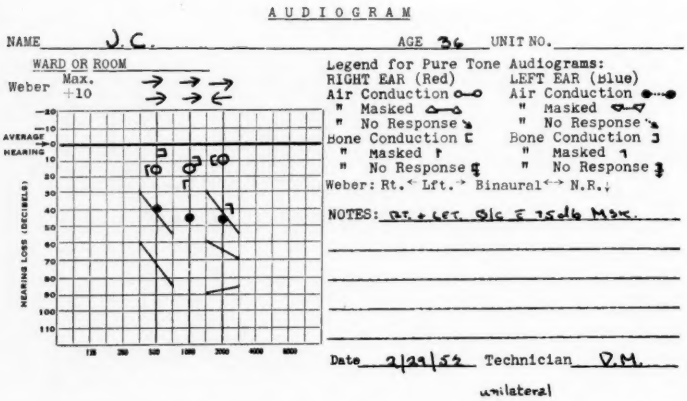


Fig. 3.—An audiogram representing a typical unilateral mixed loss. Note that, at 10 decibels above the midline threshold, the Weber lateralizes to the left ear at 500 and 1000 cps, and to the right ear at 2000 cps. Also note that, at the maximum output of the audiometer, all tones are heard in the left ear. Loudness balance confirms both a mixed loss and that over-recruitment is present at 2000 cps.

Fortunately, in otosclerosis, where bone conduction tests are most useful for determining the suitability of an ear for fenestration, it is to the poorer ear, for air conduction, that the Weber is referred. Masking the better ear, when ascertaining the bone conduction threshold of the poorer ear, is not necessary in this type of case and will, in fact, alter the very threshold which is most desired.

Experience has shown that patient-reliability for the Weber Test is not only at times inconsistent, but very often a patient will have difficulty in lateralizing to the poorer ear because his own logic tells him that it is impossible. Much care must be taken in explaining the phenomena to these people in order that satisfactory and accurate test results can be obtained.

Since it is believed that the use of masking for bone conduction measurements is hazardous, it should be restricted to those times when the test conditions warrant it. Over-masking—producing artifacts which give the appearance of a hearing loss when none is present—has been found to be very common when masking is used routinely and with disregard to lateralization.

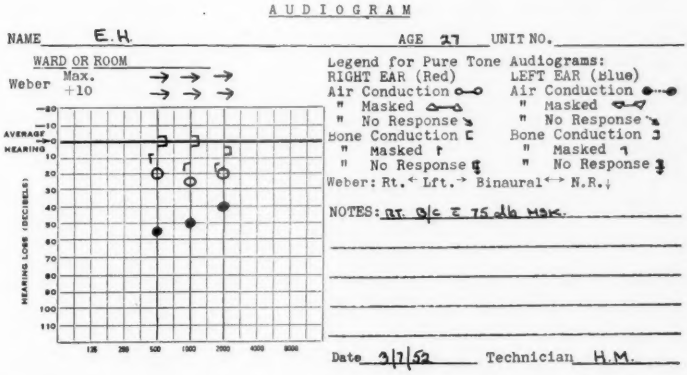


Fig. 2.—An audiogram illustrating a typical unilateral conductive loss. At 10 decibels above the midline threshold, and at the maximum output of the audiometer, the Weber is lateralized to the left, or poorer, ear. Note that the bone conduction threshold of the right ear (with masking) presents the illusion of a slight perceptive loss; this is due to the 75 decibel masking level which is effecting both ears.

On the basis of this presentation, the following procedure is recommended for routine audiograms:

1. Air conduction thresholds of the better ear,

2. Air conduction thresholds of the poorer ear—with 85 decibels of masking if there is a difference of 40 decibels or more between the air conduction thresholds of the two ears,

3. Weber Test at 10 decibels above the mid-line threshold,

4. Weber Test at maximum output of the audiometer,

5. Bone conduction thresholds of the lateralized ear—or ears,

6. Bone conduction thresholds of the nonlateralized ear—with 75 decibels of masking being presented to the lateralized ear, and

7. If further verification of the type of hearing loss is desired, the Fowler Loudness Balance Test should be used.

The combined data can then be used in the interpretation of the audiometric results (See Figure 3).

In conclusion, the "Audiometer Weber Test" has a function in the evaluation of the type and extent of hearing involvements, particularly in helping in the selection of which ears require masking. Its degree of importance, however, can only be evaluated in terms of the total information which can be obtained by audiometric techniques.

STREET ADDRESS

DISCUSSION

DR. JULIUS LEMPERT: There are many new tests suggested nowadays by lay audiologists to otologists for testing hearing. However, it seems to me that audiologists fail to comprehend that the otologist's main interest in testing hearing in those seeking to have their hearing function improved is to determine; first, the degree of cochlear nerve function that exists in a given ear; secondly, how much of this function is being utilized by the air conduction mechanism and, thirdly, how much of the existing cochlear serve function is remaining dormant because of a functionally impeded air conduction mechanism.

Judging by the number of new hearing tests being suggested by the lay audiologists it would appear that testing of hearing is a recently discovered new-born science. Perhaps if some of these audiologists would better understand the long existing otologically proven simple tests for estimating hearing function, and the ease with which these tests can be physiologically correlated to both the physical findings and clinical symptoms of ear disease, they would lose the urge for complicating and confusing the lives of the young otologists with mathematically contrived formulas for testing hearing and for predicting end results prior to the application of surgical therapy by the otologist. In order to prevent the young otologists from becoming confused I would like to remind them that despite the fact that I always encourage the search for new progressive developments in otology, the non disputable fact is still that the 30 year old audiometer, the 75 year old Weber test, the 75 year old Rinne test, and the Barany noise apparatus for masking when correctly used, have remained the best otologically proven means of testing pure physiological hearing function. The lay audiologist, on the other hand, or the otologist who prefers being an audiologist, whose main interest is not to restore physical hearing function but instead to improve the deafened person's ability to interpret the spoken voice with the use of a hearing aid and lip reading—for them the older-than-methuselah speech tests in the language used by the patient, first without and then with amplification is sufficient.

Speech tests can also be useful to the otologist interested in improving physiological hearing provided he tests the hearing for words in any language other than a language known to the patient. With masking of the non-tested ear with the Barany noise apparatus, the result thus obtained will represent pure physiological hearing function instead of part physiological hearing and part guessing ability. As an illustration of how confusing audiology can become to the otologist interested in restoring physiological hearing, I am citing a case of deafness which was referred to me for the fenestration operation.

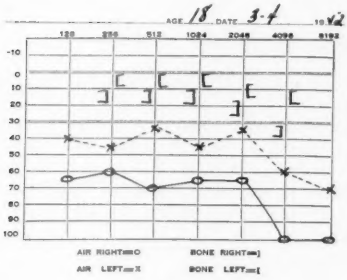


Fig. 1.

I am citing this case to point out the fallacy of accepting the findings arrived at with mathematical formulae by lay audiologists lacking in clinical knowledge and basic medical experience.

An 18 year old male with a history of progressive deafness was sent to the United States by a European otologist for me to perform the fenestration operation. The history of his deafness and physical findings in his ears permitted the tentative diagnosis of clinical otosclerosis. After diagnosing his deafness as being due most likely to otosclerosis, the next question to decide was whether or not his cochlear nerve function is still good enough to permit the restoration of practical hearing with the fenestration operation. For this purpose the following tests were made:

1) An audiometric reading was made by the lay audiologist (see audiogram, Fig. 1). As you will notice from the audiogram the bone conduction hearing in both ears appears to be still very good. The loss for air-conducted hearing is greater in the right ear. The right ear, being the worse of the two ears from the standpoint of middle ear impedance to hearing, would naturally have to be the one chosen for fenestration since the hearing by bone conduction according to the audiogram is almost equal in both ears. To check the veracity of the audiometric reading I performed personally the following tests:

2) *The Weber test*: which I personally made showed that bone conducted sound was lateralized to the left ear which is the better of the two ears. This finding was immediately considered by me as contradictory to the audiometric bone conduction findings.

Judging from the audiometrically determined bone-conduction and air-conduction findings the Weber test should have resulted in lateralization of the bone-conducted sound to the right ear which was the worse of the two ears for air-conducted sound and almost equally as good as the left ear for bone-conducted sound.

3) *The Rinne test*: I then performed the Rinne test with the steel tuning forks to check the veracity of the audiometrically recorded bone-conduction hearing for the 512-1024 and 2048 speech frequencies in the two ears and to determine whether the amount of cochlear nerve function reservoir which was not being

utilized by the functionally impeded air-conduction mechanism of the right ear which is the worse of his two ears, will be sufficient to permit the resoration of practical hearing with the fenestration operation.

To my surprise and chagrin I found that without masking he heard the tuning forks in the right ear poorly as compared to his left ear. Furthermore by masking his better hearing left ear with the Barany noise apparatus none of the three speech frequency tuning forks were heard by bone-conduction in the right ear. Judging by these findings it becaume quite obvious that the reason why the Weber test was lateralized to his left ear instead of to his right ear was the fact that the reservoir of unused cochlear nerve function was apparently greatest in his less deafened for air-borne sound left ear. Furthermore the patient's inability to hear the tuning forks by bone-conduction in the right ear with masking of the left ear completely excluded the possibility of expecting an improvement of hearing in his most deafened for air-borne sound right ear with the use of the fenestration operation.

4) *Speech tests*: Testing of his right ear with and without the use of the speaking tube revealed a complete absence of speech discrimination for both known and unknown to him language. He could not repeat simple words in any language no matter how loudly spoken into his right ear with or without the aid of the speaking tube when his better left ear was masked with the Barany noise apparatus. The patient however heard with his left ear and correctly repeated words spoken at a distance of 8 feet in a language unknown to him without the speaking tube, and at a distance of 20 feet with the speaking tube, when his worse right ear was masked with the Barany noise apparatus.

In view of my findings that there exists no cochlear nerve function reservoir in his right ear, findings which were so completely contraditory to the audiometric reading, I had no other choice but to advise the patient that the fenestration operation cannot be successfully performed upon his right ear which is the worse of his two ears and which would, under different circumstances, be the one to be chosen for surgery.

The patient and his mother were both shocked and surprised at my refusal to operate upon his right ear, since they were told by their otologist in Europe that I am to operate upon his worse of the two ears.

Understanding their plight I suggested that, since they travelled all that distance and their hopes had been so highly raised, and since his hearing in the left is no longer practical either, I would be glad to perform the fenestration operation upon his left ear wherein the cochlear nerve function reservoir is still very adequate, and his chances for obtaining a practical useful hearing improvement in that ear are still excellent.

I offered my services to attempt to give him practical hearing, which after all was what he needed and wanted, without remuneration for my services. They left both disappointed and confused and stated that they would think about it and let me know. About 10 days later I received a telephone call from a highly reputable otologist and friend of mine who wanted to know why I refused to fenestrate this patient's right ear. He stated that according to the audiometric findings of his audiology department the bone-conduction hearing in the right ear is excellent and since the air-conduction hearing in the right ear is much worse in that ear is not that the ear to be fenestrated? I informed him that my decision to refuse to fenestrate the right ear was based upon the fact that testing the patient's bone-conduction hearing with the tuning forks and masking the

RIGHT EAR							LEFT EAR						
125	250	500	1000	2000	4000	8000	125	250	500	1000	2000	4000	5000
50	65	65	55	45	60	75	25	35	40	30	35	45	70
	-15	-10	0	10	25			-10	-10	0	5	10	

Fig. 2.

non-tested ear with the Barany noise apparatus I found that he was unable to hear any of the three speech frequency forks in the right ear. My friend answered that he will call the patient back and reexamine him.

Several days later the patient returned to my office and asked to be reexamined to see if I can possibly change my opinion and also because he was very nervous and tired when I first examined him. I retested him and my findings were exactly the same, no bone conduction hearing in right ear with masking of left ear. My opinion and advice necessarily remained the same.

Several weeks later I received an inquiry about the same patient from another reputable otologist and friend of mine, with a copy of an audiogram taken in his audiology department, showing almost exactly the same excellent bone-conduction hearing in both ears as was registered by my audiometrist (see audiogram, Fig 2). He wanted to know whether it would be wiser to operate on the right or the left ear. When I told him that much to the contrary of the audiometric findings my tests of this patient with tuning forks and masking has revealed the complete absence of bone-conduction hearing in his right ear, and therefore in my opinion fenestration of his right ear cannot be considered, he said that he will reexamine the patient and hopes that we can present a united front to the mother so that she will not be so confused.

The moral of this story, in my opinion, is that the testing of hearing by the lay audiologist cannot be relied upon for diagnosing the degree of pure physiological cochlear nerve function existing in a given ear. Testing of hearing for this purpose must be done by the otologist whose basic knowledge and clinical experience with the otologic problems met with in the hard of hearing patient will prevent him from being misguided by some of the newly developed tests of lay audiologists or their erroneous use of the otologist's time-proven and well established hearing tests for pure physiological cochlear nerve function. The practice of otology is not yet that simple whereby the degree of cochlear nerve function can be estimated with the use of tests based upon mathematical formulae. Neither is the physiology of hearing as yet that well understood so as to permit the use of mathematical formulae contrived from audiometric readings of bone-conduction hearing for predicting the exact hearing improvements which will follow the performance of the Lempert Fenestra Nov-Ovalis Operation in an otosclerotic ear.

DR. EDMUND P. FOWLER, JR.: I should like respectfully to remind Dr. Lempert that any of us can be wrong sometimes with qualitative office tests and empirical hunches. Sometimes the laboratory man with his more careful quantitative tests is also wrong, but at least his data is available for check. His tests are not necessarily more complicated nor time consuming than less precise office tests. For example, the audiometric Weber test just described by Mr. Markle is quite simple and it often helps in such a case as has been described by Dr. Lempert.

The problem was one of mixed deafness with a history of bilateral mastoidectomies and allegedly a recent sudden increase in the deafness in a boy of 18 years. Incidentally, we have not done complete tests on this patient. We simply did a routine audiogram in a sound-proofed room, but as usual this included the audiometric Weber test of the type just described by Mr. Markle. This test is to our minds very useful when there is a great difference between the ears and where masking must be done for here we have the greatest trouble in measuring hearing. I am glad that he has an appointment for further testing with speech, recruitment and different types of masking for I would hate to have Dr. Lempert choke over his hat or put up a thousand dollars. I think he is quite safe for none of us really think that either ear should be operated on. When it comes to a specific rebuttal of the points brought up by Dr. Lempert I think I will let Mr. Markle tell you why he thinks Dr. Lempert might have been confused by the masking effect of a Bárány noise apparatus in the case he has just presented.

Dr. Philip E. Meltzer: I have often stated that it does not matter how many tests are performed in the evaluation of hearing. If ultimately we find these tests superfluous and uninformative, common sense will tell us to drop them. Institutions who have the facilities for making special tests should make them. Whether one uses purely clinical methods, such as fork tests, audiometer, or laboratory methods, if he has the facilities he should exercise that opportunity to the fullest. Some good will come from such reports as this.

In presenting a test of this kind, I think it is a contribution. I for one shall certainly look into its usefulness.

President Hoople: I would like to take the opportunity to discuss this paper. This presentation and Dr. Lempert's discussion of it are proof to me of the necessity of making every hearing test that we can on every patient.

I had on the American Board last week a candidate who wished an audiomertic test on the patient he was examining and I said, "It is not on the record. You have to do it with what methods are available to you." He had his own voice and he had tuning forks too.

He said, "Well, I don't know that I can do it. I need an audiometer."

I said, "Well, when you do make audiometric tests, don't you ever check them in any way?"

"Oh no," he said. (He was from a good institution too.)

I said, "How do you know your tests are correct?"

"Well, we calibrate our instrument every so often."

I make enough mistakes in my own hearing tests to know that I need to employ every available test to check my results. I feel that this test can be added to my armamentarium in addition to the audiometer and my own voice, tuning fork tests and everything else we have. Even then perhaps I would like some others.

Mr. Donald M. Markle: I would like to see if we cannot derive the answer to Dr. Lempert's question. We started out with a left ear which had a loss of approximately 45 db air conduction in the speech frequencies. A 65 db air conduction loss in the right ear.

Dr. Lempert: Yes.

MR. MARKLE: Now the Barany buzzer has an acoustic output of well over 100 db. As a matter of fact, the level far exceeds the 100 db calibrated output of the noise generators in use in our clinic and, for that reason, a balance test, for accurate measurement of the Barany's output, is quite impossible. For the purpose of this discussion, however, let's assume the Barany has an output of 100 db.

Now, if we introduce 100 db of noise into the left ear, which has a 45 db loss, it is going to hear 55 db. Am I correct? If we put in 100 it will hear 55: the difference between the noise level and the hearing loss.

At the same time, it is going to take—and I think I am being a bit conservative here—we will assume that it takes 35 db for the Barany to transfer across the head, although I feel certain that it is much less. Since the olive tip is inserted into the meatus, it is very likely that the transfer is, to a large extent, via bone conduction; this, of course, would reduce the 35 db figure considerably but, in order to prevent any misunderstanding, we will assume that the rtansfer is via air conduction and it takes 35 db to transfer to the opposite ear.

If this is so, and I feel that there is not too much question about it, the right ear is hearing the noise too, particularly if we consider this case on the basis of the conservative figures in reference to the acoustic output and transfer value of the Barany buzzer. Can you expect the ear in question to hear your voice when it, too, is being masked? Particularly if you consider the fact that your "testing voice" is probably no louder than 85 db over normal hearing?

By actual measurement, this equals approximately 100 db—which is 86 db over normal hearing (demonstrating by shouting loudly). From this, you can see that you are presenting the ear in question with only 25 db of speech—85 less the 60 db loss—and, at the same time, you are presenting masking noise to the ear under test. The masking is, in this case, masking out the signal.

DR. LEMPERT: Do you think that this patient, who does not hear by bone conduction the tuning forks in his right ear with masking of his better left ear with the Barany noise apparatus and does not hear loud spoken single words in his right ear with masking of his left better ear with the Barany noise instrument even with the use of the speaking tube in his right ear which amplifies the loud spoken words about 25-30 decibels, still can have sufficient cochlear nerve function in his right ear to permit restoration of physiological hearing with the fenestration operation?

MR. MARKLE: I am sorry, Dr. Lempert, but since I am neither a physician nor a surgeon, I cannot answer that question.

DR. LEMPERT: That is the question I am asking. The important thing is that which concerns me and the patient.

MR. MARKLE: Permit me to cite you an example. I have a bilateral conductive loss of about 50 db—otosclerotic. When the Barany is presented to either ear, with jargon being presented to the other, I am unable to repeat it. As a matter of fact, I can barely hear it.

DR. LEMPERT: If you have good cochlear nerve function you will hear me and repeat even words in a language unknown to you with the use of the Barany instrument for masking.

MR. MARKLE: That possibility has been thoroughly investigated.

DR. LEMPERT: The question we as otologists are concerned with is whether physiological hearing can be restored in a particular deafened ear.

MR. MARKLE: I realize that.

DR. LEMPERT: In order to restore to a patient practical hearing, he must have good enough cochlear nerve function to permit it.

MR. MARKLE: That is, if he cannot hear without the masking.

DR. LEMPERT: Then evidently his cochlear nerve function is not good enough to permit a restoration of practical hearing when that is the case. That is the conclusion I must reach in the above mentioned case. The lay audiologist's disadvantage is that he does not know otology. The otologist's advantage is that, although he may not know that the Barany instrument is capable of 100 decibels of sound, he knows from clinical experience that with the use of the Barany apparatus in the non-tested ear if good cochlear nerve function exists in the tested ear, bone conduction hearing in that ear is not masked out by the Barany noise apparatus. He also knows from clinical experience that when it is masked out there is not enough cochlear nerve function left to permit restoration of hearing by fenestration or any other means.

SILVER ANNIVERSARY

Arthur W. Proetz, A.B., M.D., Sc.D.

A Letter From The Publishers

Twenty-five years of outstanding success in any field surely deserves full recognition, and the publishers of the Annals of Otology and Laryngology are justly proud of our editor-in-chief, Arthur W. Proetz.

While his distinguished service on the Editorial Board has been even longer, for the past 25 years the responsibility for policy, selection of board members and maintenance of the highest scientific and production standards has rested on his shoulders.

Last year Dr. Proetz received the De Roaldes Medal of the American Laryngological Association, the highest honor available to an American otolaryngologist. On that occasion the citation read, in part:

"Endowed as are few of his colleagues with a distinct ability for research, Dr. Proetz has the gift of getting down to fundamentals. His chief interest has been nasal physiology, cytology (tissue cultures) and pathology. His flair for original investigation has borne fruit in two books which have won him worldwide renown, "The Displacement Method of Sinus Diagnosis and Treatment," published in 1931, and the unique work, "Essays on the Applied Physiology of the Nose," published in 1941. He has no peer in developing the fundamental principles of rhinology.

"A prolific writer, Dr. Proetz has contributed scores of articles on otolaryngologic subjects which have enriched the literature of his specialty, many of them dealing with phases of nasal physiology and particularly the morphology of ciliated epithelium. From his facile pen have come sections in nearly a dozen textbooks. It is fitting that one of his literary gifts should grace the office of editor. True to the traditions of the perfectionist, served with rare distinction since 1928 as Editor of the Annals of Otology, Rhinology and Larynlogy. For this service alone his colleagues owe him a debt of gratitude."

This year Dr. Proetz becomes the second American invited to give the annual Semon Lecture, at the University of London, the highest distinction offered by British laryngology.

We, who have been associated with him for so long, desire to add our gratitude for his untiring and successful achievements in behalf of the Annals. The vastly increased circulation and the fine original papers by renowned contributors from every section of the globe are tributes to the regard in which our publication is held and our Editor-in-Chief deserves the entire credit for this.

In all these years, there has never been one moment of friction, and our happy personal relations testify to his modesty and cooperation.

Regretfully, we have no honorary degrees to offer, no prizes to award, but if in the field of medical editorial work, a diploma could be awarded "Summa Cum Laude" we are sure the first recipient would be Arthur W. Proetz.

IRENE LOEB COHN

JULIAN B. COHN

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, January 7, 1952

THE PRESIDENT, DR. ARTHUR J. COOMBS, IN THE CHAIR

Differential Diagnosis of Bronchial Asthma with Special Reference To the Upper Respiratory Tract

LEON UNGER, M.D. (By Invitation)

The allergic individual may have symptoms in any part of the respiratory tract. If it affects the nose, we say the patient has hay fever or perennial allargic rhinitis. In recent years, allergic involvement of the sinuses has been included in one of these two categories. When the symptoms strike lower down, the patient has either bronchial asthma or allergic bronchitis.

The diagnosis of allergy is usually not difficult and is based on:

(1) History of allergy in the family and/or other allergic conditions in the patient.

(2) Clinical tests whereby symptoms are relieved by removal of the causative factors and increased by re-exposure.

(3) Positive skin tests which must be corroborated clinically.

(4) Symptomatic relief from such medicines as epinephrine or of the antihistaminic drugs.

(5) The finding of eosinophilia in the nasal smear or sputum, as well as in the blood.

Tonight we are reporting on four patients who were referred because of symptoms suggesting bronchial asthma. Investigation, however, has shown that each one of these patients had some other condition rather than true allergic bronchial asthma. From these examples, it is evident that the allergist must not jump to the con-

clusion that all the patients referred to him are allergic. It is also true that those who specialize in nose and throat work must always be on the lookout for patients whose symptoms result from allergy.

DISCUSSION

DR. PAUL H. HOLINGER: Dr. Unger has given us a most interesting discussion. It might be in order to ask if he would present this, or a similar paper, before the Allergy Society also. It presents clearly the correlation between wheezing and intratracheal obstruction, and certainly from the standpoint of the otolaryngologist the association of the two must be kept in mind. Very frequently an obstruction accounts for the wheeze; an overlooked foreign body and bilateral bronchial obstruction are other conditions that fall into the category mentioned. I might mention one patient who had a persistent, bilateral wheeze for approximately eight years, and finally x-rays demonstrated two large upholstery tacks, one in each bronchus.

The use of smears of sputum or bronchoscopically obtained secretions stained for eosinophiles frequently gives information as to whether the patient with bronchiectasis has a suppurative bronchial lesion alone or whether there is an allergic background. Dr. Hansel suggested that we use this diagnostic procedure, and we have found very definite clinical value to this test.

Dr. Unger mentioned the management of tracheal tumors. Very frequently endoscopic coagulation of the base of the tumor is of help, particularly if the tumor is not resectible. Otherwise, the newer forms of surgery may be helpful. Interesting work along this line is being done in other parts of the world, and recently in Honolulu we had the opportunity to see cases of Dr. Paul Gebauer, who has done resections of a portion of the tracheal wall, closing the defect by wire supported fascial grafts, and has resected stenoses with anastomosis of the distal bronchi and lung to the bronchus proximal to the stenosis, resecting the portion that had been causing wheezing for a long period of time.

I should like to thank Dr. Unger again for presenting this interesting paper to us.

DR. LEON UNGER (closing): I always enjoy hearing Dr. Holinger; he hits the nail on the head. I would have no objection to giving this paper before the Society of Allergy. I think allergists recognize their own faults, and the pendulum is beginning to swing

in the other direction. I also remember, in a discussion before the Chicago Pediatric Society, the scorn with which my talk was received. Two or three of the men, however, were interested, and it was not too long before the pediatricians were doing skin tests—some well and some poorly. It is better to do none at all than not to do them well.

It is amazing how many rare and unusual allergens there are. Within the last two weeks we have one patient sensitive to the castor bean, and one to flaxseed; we have had patients sensitive to pyragon, which is found in some wave set lotions and hair tonics, and also in linseed oil; that is why many patients have attacks while painting and decorating is being done. In Denver, asthma occurred in many children in an institution while it was being decorated, and some of it must have been due to linseed oil.

So far as foreign bodies are concerned, we always look for them and ask about them in the history, but we have had only a few such cases in our office. I was glad to hear Dr. Holinger emphasize the importance of examining bronchial smears as well as nasal. If you use them for making autogenous vaccines, the question is whether they are better than ordinary stock respiratory vaccines. I think the effects are usually non-specific.

I was interested in hs remarks about surgery. Dr. Jerome head, who removed the tumor in the case I presented, has discussed this with me, and I have left it in his hands. It seems logical, rather than have the girl have recurrences.

The Treatment of Deafness by Prosthesis

H. G. Kobrak, M.D., Ph.D.

In certain sound conduction lesions there is destruction of parts of or the whole ossicular chain. Attempts have been made to replace the missing ear drum and the destroyed ossicles by materials inserted into the tympanic cavity. The prosthesis represents a simple but frequently rather effective method of improving hearing and, in its new information may be gained as to the function of hearing. Three main groups of mechanical devices are described.

It is preferable that a natural closure of an ear drum perforation be accomplished, but if this cannot be achieved, several materials are adaptable to used as prosthesis. The prosthesis may be left in

place for several weeks without causing undue irritation. Supervision by the otologist, however, is indicated.

Any patient with a bilateral hearing defect due to destruction of the sound conducting system should be investigated as to the feasibility of a tympanal prosthesis. It is an effective therapy although its mechanism is not clearly understood. The vibratory behavior of the tympanal prosthesis can be observed during acoustic stimulation. (Motion picture demonstration) Clinical experience has shown that the prosthesis is ineffective if the middle ear cavity is completely dry, and several methods have been devised for retaining moisture within the ear. Aquaphor ointment has been used in a series of patients with good effect, in cases in which the round window niche was accessible and easily plugged. In other cases, especially in radical mastoid cavities, the "waterbag" prosthesis was used; that is, a drop of water wrapped in latex membrane. Hearing gains up to 35 db have been obtained. The prosthesis is invisible, of amazing simplicity, negligible in cost, and provided proper material is used, can be left in the middle ear for weeks or months.

DISCUSSION

DR. MARVIN TAMARI: For the record only, I would like to say that about 60 years ago Politzer suggested an artificial drum to increase hearing. He also devised an instrument which could be handled by the patient himself. We started to experiment with the audiometer, and I found in 1922 that by introduction of an artificial drum not only the hearing conduction but also the bone conduction could be improved by about 10 to 12 decibels.

DR. H. G. KOBRAK (closing): We have frequently heard that other physicians have similarly good results with a prosthesis in the middle ear, and there are reports in the literature which are promising. Nevertheless, there are quite a few patients who have lived in the Chicago area all their lives, who have suffered from chronic middle ear infection all their lives and still have not been fitted with a prosthesis. I should like to express the hope that the profession will avail itself of this simple device for improving the hearing of patients.

A Cortical Test of Auditory Function in Experimentally Deafened Cats

JOSEPH E. HIND, JR., M.D.

AND

HAROLD F. SCHUKNECHT, M.D.

(Division of Otolaryngology, University of Chicago Medical School)*

In an attempt to develop a test of function of the peripheral auditory system of the cat which would correlate well with the results of behavioral tests on the same animal, we have applied the method of evoked strychnine potentials from the auditory cortex as first described by Tunturi.** In this method, after the auditory cortex has been treated with strychnine sulfate solution, spike-like waves of electrical potential can be recorded from the cortex in response to the relatively abrupt onset of pure tones, and a determination made of the sound pressure level required for threshold at all frequencies of interest. When the average threshold values obtained for a group of normal animals are compared with those for an animal with experimentally produced hearing loss, a curve may be obtained showing the increase in threshold sound pressure in decibels as a function of frequency. The experimental results presented compare the loss of function (increase in threshold sound pressure relative to the average normal value) for behavioral and cortical tests on seven animals. Five of the animals presented a high frequency hearing loss due to lesions of the basal turn of the cochlea. The other two had partial section of the eighth nerve. The latter two cases are of hearing losses relatively more severe for the low frequencies as a result of this partial section. They are of interest in that essentially normal aural microphonic potentials were recorded from the round window in both cats, while the behavioral and cortical results indicated a substantial loss in sensitivity for the frequencies below 8000 cps. A brief indication of how the functional test results correlate with histologic findings is also presented. The behavioral test will remain, by definition, the only true test of hearing. We find that the cortical and behavioral results agree quite well. The great savings in research time offered by the cortical method cause

*This research was aided by a grant from the Veterans Administration.

**Tunturi,, A. R.: Am. J. Physiol. 162-489, 1950.

it to be a valuable tool in the experimental analysis of the peripheral auditory system.

Meeting of Monday, February 4, 1952

THE VICE-PRESIDENT, DR. NORMAN LESCHIN, IN THE CHAIR

The Treatment of Oroantral Fistulae

LINDEN J. WALLNER, M.D.

(Abstract)

For the purposes of management, alveolar fistulae may be divided into two types: the acute, of recent origin, and the chronic, of long duration.

The fundamentals of treatment of the acute case include: adequate antimicrobial therapy, preferably penicillin, intranasal drainage of the associated sinusitis, and avoidance of manipulation of the fistula. With this regimen most acute oroantral openings will close spontaneously.

In chronic alveolar fistulae the pathology consists of an epithelized tract into the antrum, a chronic infection of the maxillary sinus, usually with anaerobic organisms, and some localized osteitis about the margins of the bony opening.

The requisites for successful closure of a chronic fistula are relief of the sinus suppuration by antrotomy, combined with some type of plastic operation. This is best performed in two stages. The sinus surgery is carried out first, and when the antrum is free of infection the fistula is closed by a plastic procedure.

The commonest plastic operation is the raising and fashioning of a flap from the hard palate and rotating it over the bony opening. Berger, in 1939, described a method of forming the flap from the lateral aspect of the alveolus. An incision is made over the fistula and the edges elevated to expose the size and location of the bony opening. The epithelial lining of the fistula is removed, with any softened bone. Formation of the flap is accomplished by making two divergent incisions at right angles to the first one, and extending upward to the bucco-alveolar fold. The flap is turned outward and the periosteum is incised at the base, permitting free mobiliza-

tion by utilizing the loose bucco-alveolar fold. The flap is then secured over the bony opening without tension by means of mattress sutures, and the remaining edges are closed by single sutures. Details of the operation were illustrated by a motion picture.

The author has performed the Berger operation on ten patients with but one failure. The one unsuccessful result was in a patient who had the antrotomy and plastic operation carried out at the same sitting.

The Berger operation is readily performed. It seems especially adapted for fistulae located on the lateral aspect of the alveolus, or those situated between two remaining teeth. Wider employment of this method is advocated.

The Nasal Mucosa of Allergic Patients Before and After Treatment with Corticotropin

B. Z. RAPPAPORT, M.D.

M. SAMTER, M.D.

H. R. CATCHPOLE, M.D.

AND

S. SCHILLER, M.D.

With Technical Assistance of

C. BADER, M.D.*

(All By Invitation)

The present study is an extension of a pilot experiment begun in 1950 on the effect of ACTH in ragweed sensitive cases during the hay fever season. In the preliminary work (J. Allergy, 22:304-313 (July) 1951) nasal mucosa obtained by biopsy and stained by routine methods failed to show histologic differences in the tissues before and after ACTH therapy. In the present study there was available the mucosa of 46 patients—30 from the 1950 series and 16 from a new group seen during the August 1951 season. Of these there were 32 patients whose tissues were obtained before and after ACTH

*From the Allergy Unit and the Departments of Medicine, Pathology and Otolaryngology of the University of Illinois College of Medicine.

therapy, and 16 who received no treatment and from whom biopsies were obtained for control study. All tissues were fixed by freezing in liquid air and drying in vacuo. Paired tissues were stained simultaneously by the periodic acid-leucofuchsin method which is highly specific for substances containing carbohydrates.

The mucosa obtained at the end of four days of treatment with ACTH, when compared with that removed before treatment, showed consistently the following changes: (1) a general increase in stainability of the glycoprotein ground substance; (2) an increase in the cement substance between the epithelial cells of the mucosa; (3) an increase in the staining density and in the width of the submucous and of the perivascular "basement membrane;" (4) an increase in the number, the thickness and the stainability of the fibers in the ground substance: (5) an increase in the number and size of red-staining granules in the cytoplasm of stellate, oval and round cells present in the submucosa. These cells are believed to be undifferentiated fibroblasts (mesenchymal cells); and the granules to be glycoprotein in composition. It would appear from these results that during the allergic reaction the glycoproteins of the ground substance and the basement membranes are broken down (depolymerized) to smaller units with a proportionate loss in stainability. The effect of corticotropin is to arrest the process of depolymerization. The glycoproteins are restored toward the normal state with a resulting increase in stainability.

DISCUSSION

DR. O. E. VAN ALYEA: I have been much interested in this presentation. I know that ACTH has been used during the active stages of hay fever with reported success, at the Mayo Clinic at least. I wonder if Dr. Rappaport has formed any conclusions which will help us in a practical manner in treatment of nasal allergy of the seasonal or perennial type by the use of ACTH or cortisone. If so, would it be justifiable in routine therapy, despite the possible ill effects that have been reported from the routine use of these hormones.

DR. JOHN J. BALLENGER: Dr. Henry L. Williams has noted that there is an increased capillary permeability in allergic states. He explained this by suggesting that there was a spasm perhaps of the arterioles with subsequent anoxia and dilatation of the distal capillaries and venules. The anoxia is presumed to be instrumental in producing the increased capillary permeability and resultant edema. I wonder if Dr. Rappaport has any information to corroborate or repudiate this.

DR. WALTER THEOBALD: A year ago someone in the East reported treating certain types of rhinitis by means of a cortisone nasal spray. I would like to ask Dr. Rappaport whether he has used ACTH or cortisone in the form of a nasal spray instead of internal medication for these cases of allergic rhinitis.

DR. BEN Z. RAPPAPORT (closing): Regarding the use of ACTH and cortisone, it would seem to be an optimal means of treating an allergic reaction of short duration. Unfortunately, most allergic conditions are not of short duration; most patients are sensitive to many allergens. I have not emphasized clinical therapy. What impressed me about these people was their great satisfaction despite the small degree of improvement. I do not mean that they were not improved, but their happiness was out of proportion to the degree of improvement. There were patients with frank asthma, not severe but moderate, who said they felt wonderful.

A second point is that we are dealing with something we know too little about. If one is dealing with severe asthma that fails to respond to ordinary therapy, he should not hesitate to use ACTH. One should, however, hesitate to use ACTH for mild or moderate asthma. The temporary effect of the drug was apparent in this as well as in previous studies. ACTH was discontinued at the end of four days, and there was a rapid recurrence of symptoms within 48 hours. So far as its local use in the nose is concerned, we have had no experience with it. I have talked with those who have, and the reported results have been disappointing. I would not expect enough absorption of the material in view of the irritation and increased nasal secretion that results.

As for the reason for permeability of the blood vessels, the structural changes observed in the finer capillaries is amazing. The changes occurring in the many capillaries of the submucosa explain the rapidity of the edema. It should be noted that these changes are not limited to allergic conditions; similar changes occur in all types of inflammation. Regarding the question whether anoxia might explain the changes, one finds no evidence of anoxia. There is no vascular stasis. The rapidity of the changes (depolymerization) suggests an enzyme-like reaction. The return to normal following ACTH therapy suggests an arrest in the breakdown process permitting normal synthesis of glycoproteins (polymerization) to be reestablished.

Surgery In Intra-Oral Cancer

MAURICE F. SNITMAN, M.D.

Prior to the advent of irradiation cancer of the oral cavity was treated surgically and with comparatively poor results. The hope of increased curability following radiation therapy has not been realized. Consequently, surgery again is becoming the treatment of choice. Impetus to this modality has been provided by improvements in surgical technic and anesthesia, antibiotics, and a greater understanding of the importance of fluid and electrolytic balance. Additional measures such as parenteral and catheter feedings and tracheotomy have contributed greatly to this cause.

The histories of five patients are presented; these illustrate the variations in surgical procedure. In all cases the principle of the monobloc is adhered to. Five year cure rates are not as yet available. The improved immediate result, however, is demonstrated by absence of the almost constant excruciating pain that follows, in many instances, irradiation in one form or another. Surgical en bloc removal of the primary lesion, together with the regional lymph nodes and intervening lymph-bearing tissue, follows the fundamental principles laid down by Halsted fifty years ago when he described radical mastectomy.

DISCUSSION

DR. ROBERT LEWY: It was my privilege to scrub on a number of the cases demonstrated tonight. I certainly regard this as progressive surgery. It is a step in the right direction that our newer technics make this possible. It is a good thing that a few people are doing this type of work.

I would like to ask Dr. Snitman whether there are certain cases in which there is not a specific risk of metastases occurring to the contralateral side and, if there are such cases, were they included in this series? If such cases exist, is this amount of surgery enough, or should not a suprahyoid dissection on the opposite side be required, or perhaps even more extensive surgery on the opposite side?

DR. HANS VON LEDEN: I was very happy to hear Dr. Snitman's paper because I agree so fully with everything he has said about this so-called monobloc or commando operation as it is called. There is one additional point or commando operation as it is called. I have made it a point, in all cases in such suprahyoid nodes are posi-

tive on biopsy, to perform a radical neck dissection. If these nodes are involved I feel that there is likelihood of involvement of other nodes as well.

DR. M. F. SNITMAN (closing): These patients are not being presented as cured. The two that had seed implantation of the primary lesion should not necessarily speak for treatment along these lines. Eradication of the primary lesion of the tongue by x-ray and radium therapy is generally unsatisfactory. The patient is usually left with an irradiation fibrosis, plus the residual. The five cases operated upon are presented in the light of surgery of a similar nature in other parts of the body. It is scientifically correct, and the feeling of the men who have performed this procedure during the past few years is that there is a decided improvement over methods previously employed. So we present these patients as immediate results.

We do not perform prophylactic neck dissections on such patients since tongue and floor or mouth cancer in not a small number of cases develop crossed metastases.

There are only two kinds of neck dissection so far as nodes are concerned; one is the suprahyoid, and that is always prophylactic in conjunction with surgical excision of oral cancer. The other of course, is the total radical. When during a suprahyoid neck dissection positive metastatic nodes are encountered a total radical neck dissection is the rule.

Stenosing Peptic Esophagitis

STANTON A. FRIEDBERG, M.D.

AND

FREDERICK K. CLAYTON, M.D.

Esophagitis of mild degree is fairly common but may occasionally progress to the point where ulceration or low esophageal stenosis develop. Wilson (Laryngoscope, 61:423-447, May, 1951) recently re-emphasized this sequence in an excellent contribution. The subject has been previously analyzed by Chevalier Jackson, Allison, Benedict and Sweet, Olsen and Harrington, Vinson, Wangensteen and others.

Most benign strictures which occur at the lower end of the esophagus are probably secondary to nonspecific esophagitis. It

has been demonstrated both experimentally and clinically that esophagitis may result from the corrosive action of acid gastric secretion upon the esophageal mucosa.

Reflux of gastric secretion into the esophagus takes place when there is relaxation or incompetence of the cardiac sphincter, as in diaphragmatic hernias of the sliding type, or where partial thoracic stomach is associated with a shortened esophagus having either a congenital, spasmodic or acquired basis. Peptic ulcer diathesis, chronic alcoholism, prolonged vomiting, moribund states, indwelling gastric tubes or certain infectious diseases may also predispose to development of esophagitis.

The pathology and symptomatology of low esophageal stenosis resulting from peptic esophagitis are reviewed. Considerable responsibility rests with the endoscopist, not only in establishing the correct diagnosis but in carrying out the dilatations which control symptoms in the majority of these patients. Surgery of the esophagus and stomach offer a solution for intractable cases where failure of dilatation, recurrent hemorrhage, persistent pain or esophageal perforation complicate the clinical picture.

Case histories of six patients with stenosing peptic esophagitis illustrate the occurrence of peptic ulcer diathesis in all six; the high incidence of chronic alcoholism (five patients); hiatal hernia of the congenital type in one patient and of the acquired form in four others. An interesting sidelight was the spontaneous perforation of gastric or duodenal ulcers in two patients during symptom-free intervals between dilatations for their esophageal strictures.

DISCUSSION

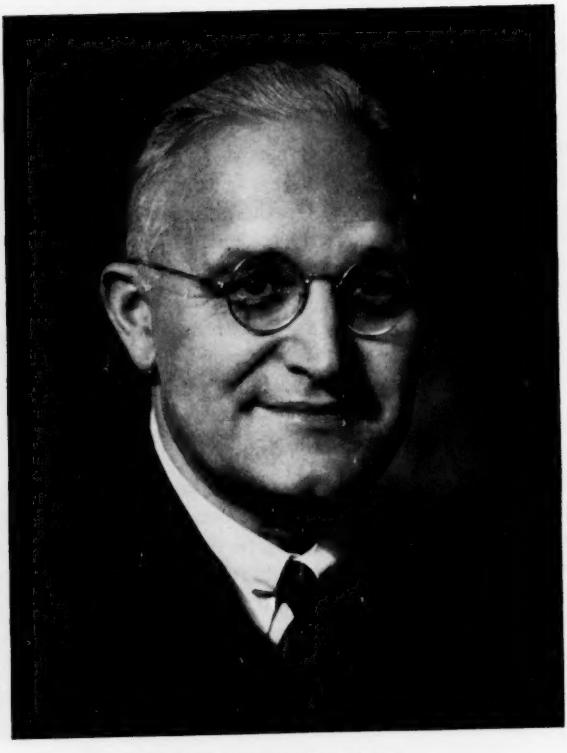
DR. PAUL H. HOLINGER: Dr. Friedberg has added another important contribution to his previous work on the esophagus and, together with Dr. Clayton, has presented an interesting group of patients. I would like to ask a question with regard to two patients: In the first patient, the stricture was so extremely high that I wonder why they classify this as a peptic ulcer of the esophagus at 27 cm from the upper teeth, and whether or not there was some other information aside from that given which caused them to consider this as a peptic ulcer. I was a little confused about the last patient with regard to the nature of his lesion; why was the plastic operation on the esophagus followed by further esophagoscopic dilatations? If further dilatations were necessary, had a stricture again developed at the point of the anastomosis, or did the reflux of secretion from the stomach cause another peptic ulcer to develop with further stenosis?

If a postmortem examination was done, what did it show with regard to the lesion in the esophagus?

DR. STANTON A. FRIEDBERG (closing): Actually, some of these patients were admitted with a tentative diagnosis of carcinoma of the esophagus and did offer diagnostic problems. When presented in sequence like this the clinical picture of esophagitis and benign stenosis becomes fairly clear, but for the sake of the record it should be emphasized that the correct diagnosis was not immediately evident in the earliest cases of the series.

In reply to Dr. Holinger's first question regarding the patient with the congenital short esophagus, it should be pointed out that a good deal of time was spent in trying to determine the etiology of his stricture. He had no severe febrile illness and no history of caustic ingestion—in fact, we practically accused him of not telling the truth about his illness. We finally concluded from biopsies obtained above and below the stricture that he did have a short esophagus, peptic esophagitis and a benign stricture. The second patient was an even greater problem. He had been operated upon so many times in the past that the surgeons were reluctant to attempt any further surgery. It was unfortunate that we were unable to obtain a postmortem examination.

Wagensteen is of the opinion that esophagitis is so commonly associated with peptic ulcer as to be considered part of the ulcer syndrome, and he feels that esophagitis may be the mildest form of the ulcer diathesis. It is true that dilatation of these strictures poses a dilemma in that, following relief of the stenosis, gastric secretion flows more readily into the esophagus. However, esophagitis is less troublesome than a persistent stricture and the final answer will doubtless depend upon actual solution of the whole ulcer problem.



MARVIN FISHER JONES
1889-1952

MARVIN FISHER JONES, M.D.

1889-1952

Marvin Fisher Jones died on May 26, 1952 at the Manhattan Eye and Ear Infirmary in New York, after a short illness.

Born in 1889, at Spencer, New York, he was the son of George Edward Jones, a merchant, and Sadie Fisher Jones. His early life was spent in Spencer. He received his A. B. Degree from the University of Buffalo in 1909, and graduated from the medical School of this University in 1913. After serving his internship at the Moses Taylor Hospital in Buffalo, New York he began the study of Oto-laryngology at the New York Post-Graduate Hospital in 1915.

At the termination of his residency in 1917 he joined the Medical Corps of the United States Army, being commissioned as a First Lieutenant. He saw service in France and Italy, and after the cessation of active hostilities was a consultant in Otology in the Army of Occupation in Germany. After his discharge from the Army he received his appointment as a member of the staff of the Manhattan Eye, Ear, and Throat Hospital in New York.

His great interest in resident teaching and his organizational ability soon became apparent by his establishing the excellent program which is now in operation. Later, he inaugurated the Conservation of Hearing Clinic which dealt with all phases of the hard-of-hearing, including the fitting of hearing aids and the educational instruction of the young.

Through the American Otological Society he established a research center for the investigation of the cochlear function and the physiology of the inner ear. This produced the technique known as aural barometry for the detection of stapedial fixation. When the fenestration operation became part of the otologist's armamentarium, he investigated the possibilities of this technique and pioneered in its introduction to his hospital.

For the past several years until the time of his death he served with distinction as Surgeon Director of Otology in this institution.

He was also formerly Professor of Clinical Oto-laryngology of the New York Post-Graduate Medical School and Hospital of Columbia University and on the staffs of many other hospitals in the environs of New York City.

His intense interest in pure research relating to otological problems stimulated him to participate in the activation of the Otological Research Council, which was fostered by the American Otological Society. In just recognition of his idealistic efforts he was elected to the presidency of the Research Council within the past year. As a further evidence of his interest in otology he was largely instrumental in establishing the Award of Merit Medal of the American Otological Society, which is bestowed by this Society from the funds of the Research Council for pre-eminent work in the advancement of the science of otology.

He was an active member of and held offices in many otolaryngologic societies, and a member of the national societies, including the American Laryngological Association, the American Otological Society, by which he was honored with the presidency in 1949, the American Laryngological, Rhinological, and Otological Society, the American Academy of Ophthalmology and Otolaryngology, and the American Board of Otolaryngology. In addition, he was a member of the American Therapeutic Society and the American College of Surgeons.

Dr. Jones was a prolific and valuable contributor to the literature on otolaryngologic subjects, particularly those pertaining to otology. He was an idealist, a zealous advocate of the truth, a ruthless antagonist of sham and deception, a man of strong convictions.

His chief outside interest was painting, in both oil and water color. As a member of the Salmagundi Club of New York since 1935, he contributed much to this institution of art. He served as Editor of the house organ for 1950 and 1951. Most significant were the outstanding honors afforded him as an 'exhibiting' member during the season of 1951. Dr. Jones was likewise an ardent fisherman.

In June 1922 he married Miss Jesse Hill, by whom he is survived with their three children James McKernon, Beatrice Clair, and Robert Marvin.

Abstracts of Current Articles

EAR

The Origin of the Perilymph.

Kley, E.: Zeitschrift für Laryngologie, Rhinologie und Otologie 30:486-502 (Nov.) 1951.

Using for these studies living and dead guinea pigs, as well as temporal bones obtained from the autopsy room, this investigator was able to demonstrate the passage of a number of different materials from the subarachnoid space through the aquaeductus cochleae into the scala tympani. Eosin, Fluorescine, a mixture of methylene blue and berlin blue, trypan blue, and a mixture of iron and ammonium citrate and potassium ferrocyanide were used. In examining sections of 28 temporal bones from guinea pigs and 20 from humans the author was not once able to demonstrate any obstructing membrane within the aquaeductus cochleae, as had been claimed by Waltner in 1948. However, in the case of humans, in order to obtain the passage of fluid through the aquaeductus cochleae from the subarachnoid space it was necessary to apply negative pressure (partial vacuum) quivalent to at least 50 mm of mercury.

After plugging the aquaeductus cochleae with bone wax and withdrawing the perilymph from the scala tympani it was found that the scala tympani refilled with perilymph, although this filling required a somewhat longer time than when the aquaeductus was not obstructed.

In order to investigate the possibility for transudation of perilymph from the blood vessels within the perilymphatic space, the author utilized guinea pigs and administered subcutaneously 2 cc of ten per cent fluorescine solution after the aquaeductus cochleae had been sealed with wax. The dye appeared in the perilymph of the scala tympani within eight to 17 minutes, in each case simultaneously appearing in the fluid of the anterior chamber of the eye. He considered this as showing that perilymph arises, at least in part, from the blood vessels within the perilymphatic space. Whether this process is accomplished by means of filtration or secretion remains to be determined. The author also used a mixture of iron and ammonium citrate one per cent and potassium ferrocyanide one per cent, which, during subsequent decalcification with hydrochloric acid, gave a precipitate of Berlin Blue within the tissues, clearly

visible in microscopic sections. In nine animals this mixture was injected into the common carotid artery. At intervals of time, from a few minutes up to three and one-fourth hours, the animals were sacrificed and the heads taken and placed in a fixing and decalcifying solution consisting of:

Formalin	40%	100 cc
Hydrochloric Acid (C.P.)	25%	200 cc
Distilled Water, q. s. ad		1000 cc

In five additional animals this iron and ammonium citrate-potassium ferrocyanide mixture was injected directly beneath the dura and the animals treated as described above. The results showed:

1. A copious precipitate of blue particles in the subarachnoid space and the ventricles.

2. An extention of the blue dye along the cranial nerves, especially along the olfactory fibers.

3. The dye impregnating the meshwork of tissue within the aquaeductus cochleae throughout its entire length.

4. A further spread of the dye from the aquaeductus cochleae into the scala tympani of the basal turn and from here, up over the helicotrema and into the scala vestibuli (observed in only one instance).

5. In one case the ligamentum spirale was diffusely covered with the stain, while in several other cases there was a concentration of dye in the scalae in front of the ligamentum spirale, that is, in the vestibular scala at the attachment of Reissner's membrane.

6. The dye was present within even the smallest vessels of the ligamentum spirale and in the vena spiralis, both within the lumen and in the tissue surrounding this vein. This was interpreted by the author as indicating a possibility that the ligamentum spirale may be the organ of resorption for the perilymph.

7. In one case the blue stain was found in the perilymphatic spaces of the vestibule and semicircular canals, both within the lumina, and within and surrounding small blood vessels in the supporting framework of the perilymphatic space.

8. In not one single instance was any of the dye found within the ductus cochlearis or any of the endolymphatic spaces of the labyrinth.

9. There was a spread of the Berlin blue in the internal auditory canal, in the modiolus, along the nerve fibers and blood vessels

of the spiral ganglion, through the Lamina spiralis ossea, out to the crista spiralis of the basilar membrane and the organ of Corti. In the modiolus the dye was not only perivascular and perineural in location, but also within the lumina of the smallest vessels within this area. This was considered significant as a possible source of perilymph or as a possible focus for resorption.

The author concludes on the basis of these studies that the aquaeductus cochleae is open for the passage of fluids and of substances of low molecular weight, and probably for colloids and particulate matter. The direction of flow is from the subarachnoid space toward the inner ear. The aquaeductus cochleae, in man, while patent, does present a considerable degree of resistance to the passage of fluids. There appear to be other sources of perilymph, in addition to that which comes through the aquaeductus cochleae from the subarachnoid space. The possibilities comprise origin from the endolymphatic spaces (as presented by the work of Max Meyer in 1951), origin from the capillaries in the walls and trabeculae of the perilymphatic spaces, and origin by spread from the sub-arachnoid space along perineural and perivascular spaces in the internal auditory and the modiolus.

For anyone interested in this subject, this article has a full bibliography of recent papers, both from Europe and America.
aged fifty-one at the time of death.

HARKINS.

Histologic Study of an Operated Case of Otosclerosis Three Years after Fenestration.

Nylen, B.: Acta Societatis Medicorum Upsaliensis 56:101-107 (Sept. 29) 1951.

A histologic study has been made of the temporal bones of a woman operated on for otosclerosis. A fenestration on the lateral semicircular canal of the more severely affected ear resulted in an immediate improvement in hearing, which, however, rapidly regressed. Re-operation four and one half months later resulted in no real improvement. At the second operation the fistula was found to be closed. The patient died three years later of an unrelated disease, aged fifty-one at the time of death.

Histologic examination showed an extensive otosclerotic process, bilateral, symmetrical and active, with ankylosis of the stapes on both sides. The labyrinthine fistula was entirely overgrown with osteoid bone. Otosclerotic changes, mainly of the quiescent type,

were found in the vicinity of the site of operation. Microphotographs are presented.

The author describes briefly five other reported cases from the literature in which histologic studies had been made of temporal bones of otosclerotic persons, previously subjected to fenestration, who died later of some unrelated disease.

HARKINS.

The Preservation and Restoration of Hearing.

Cawthorne, T.: Acta Oto-Laryngologica 41:1-8, 1952.

This paper sketches the changes which have taken place in otology in the last few years, so that now the main interest is in the preservation and restoration of hearing. These changes may be attributed to developments in electronics resulting in scientific audiometry and greatly improved hearing aids, the influence of chemotherapy and antibiotics on suppurative disease and the development of nonsuppurative temporal bone surgery. In order to meet these changes the otologist not only must learn new methods of examination and of technique but he must make use of the knowledge and skills of workers in allied fields of electronics, acoustics, physiology, psychology, etc.

HILL.

Principles and Basic Techniques of Adult Auditory Rehabilitation.

Bergmen, M.: Acta Oto-Laryngologica 40:324-335, 1951-52.

Auditory rehabilitation is concerned with the readjustment of the whole person. In addition to the restoration of communicative effectiveness the patient should be psychologically rehabilitated. There seems to be no distinctive psychology of deafness but loss of hearing tends to emphasize certain personality aspects peculiar to the individual. This problem demands the combined efforts of a "rehabilitation team" capable of dealing with all its phases.

HILL.

Special Methods of Audiological Training for Adults.

Birgmen, M.: Acta Oto-Laryngologica 40:336-345, 1951-52.

This well-written article brings out the fact that auditory rehabilitation demands more than the use of a hearing aid, that it

"does not alone reverse the profound personality changes which accompany even a moderate loss of hearing." Auditory training includes developing discrimination of speech in noise, efficient listening habits, understanding of the hearing aid as an instrument, discrimination of speech sounds and tolerance for loud sounds. The importance of a speech training program is demonstrated by examples of voice and articulation changes typical of different forms of deafness.

Speech reading is of value to all suffering from a hearing loss, being of considerable assistance even to those who do well with a hearing aid, and is the only hope for the profoundly deaf. The goal of any training program should be to equip "the individual to meet his vocational and social needs as quickly and as completely as possible within his ability."

HILL.

Audiologic Assessment of Results in the Improved Fenestration Operation.

Shambaugh, Jr., G. E.: Acta Oto-Laryngologica 41:43-48, 1952.

Both the final level of hearing and the decibel gain are significant in assessing the results of the fenestration operation. An analysis of the cases studied at Northwestern University showed 75 to 80% of cases of otosclerosis without significant nerve involvement reached the level of practical hearing in one year. Of those with early nerve degeneration similar improvement was found in 40%, while in the borderline cases with definite nerve involvement only 10% achieved this; although 75% of these showed a significant gain in terms of decibels. A method of comparing the results of the use of a hearing aid with that following fenestration is described. In the majority of cases the hearing after operation equalled that obtained for speech with a hearing aid and surpassed it for discrimination.

HILL.

Psychophysics of Hearing.

Hirsh, I.: Acta Oto-Laryngologica 40:273-282, 1951-52.

The development of psychophysics in its relation to Otology is discussed in detail. Electro-acoustic technics have made possible norms for the absolute thresholds for pure tones and for intelligibility for speech. The understanding and cooperation of the otologist

in this work can result in better methods of audiometry and in the more effective employment of amplification, education, and surgery for the benefit of the deafened.

HILL.

Phychogenic Deafness (Emotional Deafness).

Blegvad, N. Rh.: Acta Oto-Laryngologica 40:283-295, 1951-52.

This paper is based upon the experiences from World War I and II and summarizes the views of numerous investigators, dealing with acoustic trauma. In the first period, before the advent of audiometry, it was assumed that the deafness was a result of organic changes in the labyrinth, based upon the findings in the ears of experimental animals subjected to intense acoustic trauma. More recent investigators have doubted the validity of the assumption as the labyrinths of these animals are more delicate and sensitive than those of man. It is now recognized that generally the loss of hearing is ½ to 1 octave above the frequencies of the provoking sound. Many authorities feel that there is an organic basis for hearing loss in these cases together with a functional overlay but that the organic loss is mild or only temporary. Others consider the loss entirely functional.

The diagnosis depends upon the mental attitude of the patient, variation and discrepancy in various tests for hearing, easily acquired lip-reading or "unconscious hearing," together with the use of the Doerfler-Stewart and psychogalvanic tests, among others. Once the diagnosis is established psychotherapy is indicated.

HILL.

The Recruitment Factor in Hearing Tests.

Huizing, H. C.: Acta Oto-Laryngologica 40:297-305, 1951-52.

Hearing impairment may be considered as conductve in type when the loudness perception remains normal regardless of whether the lesion is in the middle ear or in the cochlea. If the loudness function is normal, normal loudness patterns can be restored by reducing the distance of the sound source or by amplification. If the loudness function is abnormal there results a distortion with a discrimination loss involving speech intelligibility. Recruitment or abnormally rapid increased loudness perception may be encountered in cases of nerve deafness, such as Meniere's disease, acoustic trauma, lues, acoustic neuroma, meningitis, etc. in which amplification generally is futile.

HILL.

Instruments and Materials for Speech Audiometry.

Carhart, R.: Acta Oto-Laryngologica 40:313-323, 1951-52.

The advantages and disadvantages of monitored live voice and recorded presentations are discussed. Basically the former is more flexible while the latter can be more accurately standardized. Monitoring is necessary with both methods. Either headphones or a loudspeaker may be employed. When determining the efficiency of a hearing aid the latter, whereby the patient is tested in a sound field, is preferable. A speech audiometer should be of high quality and capable of covering a wide range of presentation levels. For clinical work calibration on the basis of thresholds for speech for normal ears is adequate. The spondee word list is used for threshold tests and the PB list for discrimination.

HILL.

Hearing and Deafness.

Cawthorne, T.: Acta Oto-Laryngologica 40:257-263, 1951-52.

This is a delightfully written article in which the author considers the phylogenesis of hearing, from the involuntary stimulus with its warning of approaching danger and its employment as a secondary sex characteristic, to its ultimate development in Man with its use in communication. While noting the lack of symptoms for those afflicted with deafness and its effect upon them, he shows how the individual can overcome this handicap by referring to the example of Beethoven. While this is an old device, so frequently employed as to be almost hackneyed, the author's treatment is such as to command interest and appeal.

In considering the causes of deafness he paraphrases Shakespeare's Seven Ages of Men most effectively, pointing out the common etiological factors resulting in hearing loss from infancy to senility and suggesting what may be done for each. He closes with the statement, "The challenge of deafness has been joined and is well on the way to being defeated."

HILL.

The Physiology of the Hearing Organ in the Light of Recent Data.

Bijtel, dr. J.: Acta Oto-Laryngologica 40:265-272, 1951-52.

The author states that our present-day theories of hearing fail to explain the physiological process involved. He points out the limitations of electro-physiological studies due to the anatomical situation

of the acoustic nerve and feels that too much emphasis has been placed upon mechanistic principles and that consideration of hearing as a psychic process is necessary.

This article should be of interest to all otologists as well as workers in the field of audiology.

HILL.

NOSE

The Diagnosis of Intranasal Cephalocele.

Fleischer, K.: Zeitschrift für Laryngologie, Rhinologie und Otologie 30:466-469 (Oct.) 1951.

Intranasal cephalocele is not a common condition, only 28 cases having been reported in the literature. The author adds a report of one case of his own, occurring in a 14-month old infant. This child presented a cherry-sized mass in the right nasal passageway; it was firm, elastic and covered with smooth, pale mucosa. Medially the mucosa appeared to continue on to the nasal septum. A biopsy of the mass had been performed elsewhere and the specimen reported as showing an inflammatory reaction in young connective tissue. The author condemns this as a dangerous procedure. The infant showed broadening of the root of the nose and an increase in the distance between the inner canthi of the eyes. X-ray examination revealed broadening of the nasal portion of the frontal bone. These points were useful in making the diagnosis. The tumor was punctured with a needle, using aseptic precautions; no fluid was obtained. Operative removal was deferred until the child was older.

In discussing this condition the author states that these are congenital defects, the cause of which is not known. In only five of the twenty-nine cases reported was the correct diagnosis made prior to removal with snare, or biopsy by means of a punch. These procedures had been done on the mistaken assumption that a nasal polyp or a neoplasm was present. Such procedures are dangerous: one can expect cerebrospinal rhinorrhea and meningitis to follow.

Five factors are listed which point to the correct diagnosis:

1. The early age of the patients—all of these cases occurred in infants or very young children.

2. Broadening of the root of the nose and an increased distance between the inner canthi of the eyes.

3. X-ray evidence of a bony defect is rarely demonstrable, but broadening of the frontal bone in its nasal portion is significant.

4. A smooth, firm, elastic and slightly compressible intranasal mass is found. Characteristically, the mucosa in its upper part appears to go without interruption on to the septum. Needle puncture of the mass may or may not strike cerebrospinal fluid.

5. An awareness on the part of the physician of the possibility of such a defect is considered the most important single factor.

As to treatment, surgical removal by an intracranial approach, with repair of the bony defect is the only treatment. It is advisable to defer operation until the infant is several years older.

HARKINS.

The So-Called Gangrenous Rhinitis,

Miura, Schinichi, Otolaryngology (Toyko) 24:283-328 (July 20) 1952.

Miura reports a case of gangrenous rhinitis in a man aged 50, which in the beginning had the appearance of being a reticulosarcoma. But further clinical observation and microscopic studies made of the tissue after healing had taken place rendered the affection to be considered as so-called gangrenous rhinitis. Penicillin in this case had no effect whatever but with radium a complete healing was obtained. For two years, no recurrence has been observed.

HARA.

LARYNX

Empyema of a Laryngocele.

Bachmann, K. D.: Zeitschrift für Laryngologie Rhinologie und Otologie 31:19-24 (Jan.) 1952.

One case is reported of a 25 year old woman who first noted a painful swelling in the left side of her neck one and one half years previously. It had been incised by the local medical doctor and pus evacuated; shortly thereafter she coughed up pus and blood. Then she remained well, except for a small lump in the left side of her neck, up until three weeks before admission to the University Clinic at Marburg. At that time the swelling on the left side of her neck began to enlarge. Fever and dyspnea developed. Prior to this time she had never had any difficulty with speech, swallowing or respira-

tion. During her journey to the Clinic the dyspnea became alarming and she was brought in to the clinic gasping for air. Examination revealed a marked swelling of the epiglottis with displacement to the right by an apparent swelling on the left side. The upper entrance to the larynx was almost completely closed by this swelling; the vocal cords were not visible. There was a swelling of the entire left side of the neck, extending from the base of the tongue to the clavicle, with a fluctuant mass within this swelling as large as a hen's egg. During the examination respirations stopped; an endotracheal tube was introduced, artificial respiration given and stimulants administered. In spite of these measures the patient expired from asphyxia.

Post-mortem examination showed a cyst, lined with pseudostratified, ciliated epithelium, presenting between the hyoid bone and the thyroid cartilage on the left side; it was filled with pus. Apparently, the cyst had previously been connected to the ventricle of Morgagni by a long narrow tract, now obliterated by connective tissue, although remnants of the tract remained. Infection had spread from the cyst out into the soft tissues of the neck, which showed massive, brawny edema. The laryngeal airway was closed by the cyst and the swollen tissues around it.

Reports of such cases are rare. The author was only able to find two reports of similar cases in the literature.

HARKINS.

Contribution to the Problem of Decannulation in Infants.

Sercer, A.: *Revue de Laryngologie, Otologie, Rhinologie* 73:60-65 (Jan.-Feb.) 1952.

The author calls attention to the tendency for stenosis to occur in the case of infants who have worn a tracheal cannula for a long period of time. He demonstrates the action of the postero-superior convex wall of the cannula in pressing the soft anterior wall of the trachea inward as it passes into the tracheal lumen. He also points out the tendency for the lower end of the cannula to traumatize the tracheal mucosa and stimulate the formation of granulation tissue at this site. He attributes this action, at least in part, to the guard or flange, which holds the cannula to the infant's neck. The comparatively large size of this guard on the commonly used tracheotomy tube causes the entire tube to be moved about repeatedly by movements of the infant's neck.

The author's method of decannulation consists, first, of revising the tracheotomy surgically. The compressed portion of the tracheal wall is elevated and sutured to the skin. Following this, a very short, narrow cannula is introduced. This new cannula has no inner tube; it has an extremely small guard, fastened by a silk cord around the infant's neck. The cannula is almost straight, having only a slight downward curvature. It barely penetrates the anterior wall of the trachea. These special short cannulae were made up individually to suit each case, by cutting down the inner tubes of standard cannulae. These short cannulae do not deform the trachea by pressure, do not traumatize the mucosa nor cause the formation of granulation tissue within the tracheal lumen. The extremely small guard allows the cannula to remain stationary when the infant moves his neck, thus avoiding trauma to the tracheal mucosa. The cannula is changed as often as necessary. After several days of wearing this special short cannula, during which the infant is under the constant supervision of a nurse skilled in tracheotomy aftercare, decannulation is completed. During the last four years the author has applied this technic in 12 infants, with complete success in all cases.

HARKINS.

MISCELLANEOUS

A Case of a Voluminous Cervical Angioma.

Mesnage, J., and Portmann, M.: Revue de Laryngologie, Otologie, Rhinologie 72:753-754 (Nov.-Dec.) 1951.

A single case is reported of a cervical tumor occurring in a seven year old girl. The neoplasm was the size of an orange, somewhat lobulated, encapsulated and firmly attached to the common carotid artery and internal jugular vein. The tumor was continuous at its superior pole with the internal jugular vein. It was dissected without difficulty. Microscopically, the lesion was interesting in that it presented the picture of an angioma, with numerous vascular spaces, apparently developed within active lymphoid tissue. There were numerous germinal centers in the tissue between the vascular spaces, which showed lymphocytes in all stages of maturity. The authors are not able to decide whether the angioma originated within a lymph node, or whether it invaded a lymph node from without.

HARKINS.

Speech Audiometry in Clinical Evaluation.

Carhart, R.: Acta Oto-Laryngologica 41:18-42, 1952.

While tests on large groups show a correspondence between speech and pure tone audiometry this may not hold in the individual case. When a discrepancy between the tests is found the reasons for it must be sought. This may indicate a psychogenic factor or central neurological damage, speech discrimination difficulties or phonemic regression, possibly due to central vascular changes. The importance of speech audiometry in determining the indications for, and the predicted effectiveness of a hearing aid is illustrated by several case reports.

Speech audiometry adds to the "understanding of the practical limitations faced by the patient because of his hearing loss."

HILL.

Books Received

Diseases of the Nose, Throat and Ear.

By Hall, I. Simson, Surgeon to the Royal Infirmary, Pp. 463, Fifth Edition, with 8 colored plates, Baltimore, Maryland, The Williams and Wilkins Company, 1952. (Price $4.00)

Der Gesunde Und Der Kranke Kehlkopf Im Röntgenbild.

By Griebel, Prof. Dr. Med. C. R., Pp. 170, with 156 illustrations, Stuttgart, Georg Thieme, 1952.

Die Krankheiten der Nasennebenhöhlen, der Ohren und des Halses im Röntgenbild.

By Mittermaier, Prof. Dr. R., Marburg/Lahn, 2nd Edition, Pp. 232 with 483 illustrations. Stuttgart, Germany, Georg Thieme, 1952. (Price DM 66)

Acta Oto-Laryngologica, Supplementum 102, On the Treatment of Corrosive Lesions in the Oesophagus, An Experimental Study.

Krey, Holger, Pp. 49, Royal Academic Hospital, Uppsala, Sweden, 1952.

Leitfaden Der Praktischen Audiometrie.

By Langenbeck, Prof. Dr. Bernard, Pp. 156, with 37 illustrations, Stuttgart, Georg Thieme, 1952.

Notices

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations:

October 6-10, 1952, in Chicago, Illinois, at the Palmer House.

April 21-25, 1953, in New Orleans, La., at Roosevelt Hotel.

DEAN M. LIERLE, M.D., Secy.
University of Iowa
Iowa City, Iowa

UNIVERSITY OF ILLINOIS

For the period of the National Emergency, the University of Illinois College of Medicine will offer a combined three year residency training program in Otolaryngology, which will include the basic course material, in fulfillment of Board requirements.

Residents will rotate through the Research and Educational Hospitals, the Illinois Eye and Ear Infirmary, the Hines Veterans Administration Hospital and the various affiliated institutions. The residency will be so flexible that should it be interrupted because of military service, the period of training may be resumed upon returning to civilian life.

Under this arrangement, no course fee is to be involved and in the case of most of the aforementioned institutions, a stipend is provided.

For further information, kindly address: Head of the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.